105
002
CUK-H00704-28-P16253

BRAIN

A JOURNAL OF NEUROLOGY

28

UNIVERSITY OF CALCUTTA
LIBRARY
ADVANCEMENT OF LEARNING

EDITED
FOR THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM

BY

HENRY HEAD, M.D., F.R.S.

Assisted in the selection of Papers by

SIR JOHN BATTY TUKE, M.D., LL.D.

H. C. BASTIAN, M.D., F.R.S.	C. A. MERCIER, M.B.
JUDSON S. BURY, M.D.	W. JULIUS MICKLE, M.
W. D. HALLIBURTON, M.D., F.R.S.	J. S. RISIEN RUSSELL,
SIR VICTOR HORSLEY, F.R.S.	ERNEST H. STARLING, M.D., F.R.S.

VOL. XXVIII.

London:
MACMILLAN AND CO., LIMITED
ST. MARTIN'S STREET, W.C.
NEW YORK: THE MACMILLAN COMPANY
1905

LIBRARY

BRAIN.

PART I., 1905.

Original Articles and Clinical Cases.

THE RELATION OF THE LUNACY LAWS TO THE TREATMENT OF INSANITY.

BY SIR JOHN BATTY TUKE, M.D., LL.D., M.P.

Presidential Address Delivered before the Neurological Society of the United Kingdom on February 16, 1905.

THE object of this address is to present to the Society as succinctly as possible the relation of the law to the treatment of the insanities, and the disabilities under which we, as a profession, lie in this respect. I feel no apology is needed to the Neurological Society of Great Britain and Ireland for undertaking this task, for I am never tired of quoting the words of Griesinger uttered nearly forty years ago, when he said, "Diseases of the nervous system form one inseparable whole of which the so-called mental diseases only embrace a certain moderate proportion." And I am further supported by the feeling that the vast additions to our knowledge of the structure and function of the nervous system, contributed by members of this Society, have no small bearing on the political position.

I am aware the subject has been under the consideration of certain of our members in one of its aspects, but I propose to regard it from the broadest standpoint, and deal with it as it affects the physician, the patient, and the public. It is a question which has occupied my thoughts for many years. I have written about it and spoken about it, but I cannot resist the temptation to avail myself of this exceptional opportunity to reiterate my views, notwithstanding that I shall be subject to the charge of repetition. The questions involved are of such national importance, that I must bow to the criticism if it be advanced.

I believe I am justified in stating that there is a widespread desire for improvements and modifications of the existing Lunacy Laws. This feeling holds with the medical profession for professional reasons, with the public on account of fiscal considerations. We have to deal mainly with the first set of reasons, although in order to buttress them, in order to make them efficacious, we may have to collate them with the second.

My primary contention in support of the demand for reform is that an important and steadily increasing change has come over our conception of so-called mental disease. It would be futile on the present occasion to review in support of this contention the history of the treatment of the insane during the barbaric periods prior to 1800; but it must be remembered that the dregs of the revelations brought to light by various inquiries instituted about that time still exercise some influence on public opinion. The memory of old scandals dies hard. During what may be termed the humanitarian period which may be roughly computed as existing during the first fifty or sixty years of the last century, the study of insanity occupied the attention of many physicians. For the most part, however, their study proceeded on psychological lines, and the prominence of the mental symptoms tended to blur the main issue. It cannot be denied, in fact, it should be proudly asserted, that during this period eminent clinicians arose, of whom Calmeil may be held as the type and the chief, who demonstrated the association of mental symptoms with certain bodily conditions, and who established clinical groups, certain of which still hold ground. Yet proof of the hold maintained by the psychological conception on the medical mind is afforded by the fact that in the "Nomenclature of Diseases" prepared by the Royal College of Physicians of London, and issued by the Registrar General in 1863, all the insanities were comprised under *six* "Disorders of the Intellect." That such should be the trend of professional and public opinion was inevitable in the then existing state of knowledge.

I have no hesitation in expressing the opinion that during the last forty years a vast change has come over the views of the profession in respect of the insanities, and that that change is due to our extended knowledge of the structure and function of the nervous system acquired during that period, and to the connotation of such knowledge with the advances in general medicine. I am not going to labour this argument by a recapitulation of the various acquisitions of science. I would rather put it this way—I would ask those who are younger amongst us to try to realise what their mental attitude towards such a class of symptoms as characterise the insanities would have been had they lived at a time when the naked-eye topography of the convolutions was undescribed, when nerve tracts were for the most part undifferentiated, when localisation of function of cortical areas was not evolved, when only some two or three men in Great Britain could demonstrate a nerve-cell, and that imperfectly, when the microscope could not be brought to bear on the morbid conditions of the nervous tissues, when, in fact, the very institutes of medicine on which the physician now relies when forming his estimate of the nature of a case were entirely wanting. I do not know what the student of abstract philosophy may think, but I hold that, along with the elaboration of an apparatus, along with the demonstration of the paths of functional activity, a higher general conception of the nature of morbid mental symptoms has been arrived at than was possible when all we had to depend on was vague induction. It would be a work of supererogation, even if time permitted, to follow out this thesis in detail, for I hope I carry the general sense of the meeting with me.

But I desire to indicate what I consider to be the point on which our acquisitions concentrate themselves in respect of the insanities. We relate in our minds the existence of a delicate and complex apparatus, with definite proof of its susceptibility to the rapid action of disease and with distinct evidence of its power of regeneration. The theory of the dissociation of neuron groups is not merely an inference applied as an explanation of the mental phenomena of certain forms of insanity, but up to a point it is a proved scientific fact. At what part or parts of the apparatus the interruption occurs is subject for further enquiry, but, if we may conjecture from the rapid results of research during the last few years, we may look hopefully to a not very remote future for important developments bearing on physiology, and, consequently, on medical psychology. Further, physiological observation has succeeded in breaking down the old idea of the action of the mind on the body. There is a wide difference between the conception of the action of the *organ* of mind on the body and the popular idea that the mind acts directly on other organs. We no longer, for instance, regard failures of function of the abdominal viscera in the insane as due to mental action, but as due to the impairment of the cerebral trophesial function which occurs coincidently with the appearance of mental symptoms.

Again, further, our whole conception of the nature and causes of acute insanity has been modified, I might almost say revolutionised, by the gradual advance in our knowledge of the action of toxins upon the structure and functions of the nerve-cell, and especially of the cortical cell. There is now the strongest evidence of toxæmia in certain forms of acute insanity and of the rapid implication of structure as a result of these poisonous agents. We are being forced to the conclusion that the mental symptoms in the acute psychoses are manifestations of a toxic physical condition, just as the delirium of fever is a symptom of certain phases of intoxication of the nervous system.

In fact, I may say that there is no development of medicine which has not been brought to bear on the subject, and with important results.

Granted what has been said as to changed views on insanity, the question arises what bearing has that change had on the treatment of the various conditions comprised under that generic term. I hold that it emphasises and gives reason for the belief that the insanities must be submitted to treatment in their earliest stages, reasons which can be adduced before the legislature. This, of course, is true of all diseases, but in regard to the insanities the position is complicated by the false conception of their nature, still to some extent existent, and accordingly the public and the legislature do not yet regard them as the symptoms of corporeal disease. Rough asylum statistics showing the relation of recoveries to the period of incidence of symptoms have not carried conviction to the general public, and accordingly the tenor of our laws is towards the *protection* of those mentally afflicted rather than towards measures calculated to procure the cure of sick persons.

It is on these general grounds that I have urged on the Government the appointment of a Royal Commission to enquire into the subject in all its relations, and to consider whether a change in the Lunacy Laws is or is not demanded in the public interest.

The law under which we now work in England is a strictly lawyer's act. It was constructed without reference to any medical authority whatever. Neither medical Commissioner in Lunacy, nor Chancery Visitor was consulted. The leading idea was the protection of the public and the lunatic. No provision was made for the early treatment of patients. There was certainly allowed the certificate of emergency, but this was grudgingly admitted, and in at least one, if not two, Lunacy Amendment Bills, it was proposed that the term should be shortened. About the time the main Bill was introduced there had been one or two "scares" which had been most unjustifiably "written up" by certain papers. As I have often said the Bill was conceived in suspicion and born in panic. It contained one good provision and one ridiculous one, but taken over it all it was bound together by such an amount of hampering red tape, that its action has been found cumbrous and irritating. The clause I have designated as ridiculous is the one by which a person certified to be insane by two medical practitioners and committed to an asylum, may be submitted to the opinion of a justice of the peace, which determines the position. To this clause we may apply the *ex pede* line of argument, for it gives expression to the feeling of suspicion which permeates the whole Act. It was asserted by the promoters of the Bill that it was founded on the Scottish system. It did so, so far as the method of certification was concerned, but it took no account of two of the most important features of the Scottish Acts, viz., adequate power of inspection, and liberty to medical practitioners to treat incipient and early cases without certification. I have argued elsewhere that the liberty granted to us in the North cannot be accorded to a country in which the inspecting power is ludicrously insufficient. I am not going to trouble you with statistics, but will merely state that in Scotland that power is more than ten times greater than in England. As a consequence there is in that country a feeling of security in the public mind, without which we could not exercise the power conferred on us. As to its working, all I can say is that Clause 13 of the Scottish Act of 1866, by which we can treat cases in private houses for six months under a certificate of a medical person, and without reporting to the Commissioners, has worked smoothly for forty years, and that in no single instance has the slightest suspicion of abuse arisen.

The first step towards lunacy reform in England is a considerable increase in the number of Commissioners. The English Commissioners, in their last report, urge this strongly on the Government, in order that they may be able to perform adequately the ordinary duties of the office. I urged this point as strongly as I could when speaking on the Lunacy estimates, and pressed hard for the appointment of a commission to deal with the whole matter. The Attorney-General in his reply gave me no hope, and appeared to be satisfied with existing conditions. Within a week, however, he introduced a small Bill which proposed to grant to England a modification of Clause 13 of the Scottish Act of 1866. But it avoided the main issue, and it would have been my duty to have opposed the Bill if it had reached the second reading stage—which it did not—on the ground that it was merely a sop to Cerberus.

I want, however, to take up a much broader issue. The Scottish clause I have been speaking of mainly applies to the monied classes. What are we to do for the poor and for those of moderate means? We all know that incipient cases derived from this class cannot be submitted to treatment till symptoms are so pronounced as to warrant certification, that is to say, till such time as morbid action has made such advance as to retard recovery or prevent it altogether. *To the accumulation of unrecovered insane persons* the numerical increase of lunatics (I use the technical term) is mainly due. It is not entirely due to this cause, for there are others, such as the Government grant, the consideration of which can hardly be brought within the scope of this address. I know I can appeal to the experience of many here present that recovery is not difficult to obtain if treatment is applied during the initial stage, and that such treatment is most satisfactory in a large proportion of cases, inasmuch as not only is recovery from the immediate attack affected, but the tendency to recurrence is weakened. In fact, the cure often becomes complete, in that morbid action on the tissues having been arrested, and full opportunity having been given for their regeneration, no permanent solution of continuity remains.

Can analogous methods of treatment be provided for the insane of the poorer classes? That it can, in the opinion of those best able to judge, is amply set forth in the report of the committee of the London County Council appointed in 1890, at the instance of Mr. Brudenell Carter, to consider the propriety of erecting a hospital for the insane. The principal finding of the committee, founded on "the opinion of the most eminent and most experienced members of the medical profession," was to the effect that it was desirable that "an adequately equipped hospital, containing 100 beds, for the study and curative treatment of insanity, should be established in the Metropolis." Why and wherefore, and under whose influence this modest scheme was not carried into effect, I do not care to consider at present. As a matter of fact the report was pigeon-holed, and has not affected in any way the policy of the Council, except to the extent that or two exceptions they were evidently drawn from the poorest class, such as one finds in pauper asylums. Of these one-fourth were suffering from alcoholism in one or other of its phases; one man was convalescent from violent alcoholic mania; the remainder were the subjects of more or less intense delirium, whose treatment in an asylum or general hospital would have been necessary had the reception wards not been in existence. I emphasise this fact because it has been loosely stated that these wards are chiefly devoted to "drunks." That is not so, for of the total number admitted since 1890, only 28 per cent. were alcoholics. The symptoms of the other cases in hospital at the dates of my visits were referred to the usual classes of insanity as set forth in asylum reports. The average length of residence is twenty-five days, but it may be extended to six weeks. In a word, the class of cases was, allowing for social position, of the same type as we meet with in private practice, and which we treat either to a successful termination at their own homes, or in private dwellings, or relegate to asylums if unfitted for such treatment. This hospital provides for the poor a method of early curative treatment, which elsewhere can only be vouchsafed to the rich. I have gone thus far into details in order to show that the feasibility of a system by which the curative treatment of the insane poor during the earlier stage of the condition has been reduced to demonstration. It is the only institution of the kind in Great Britain. The only subject for regret is that it is not so fully utilised as it should be as a clinique for the students of the Medical School of Glasgow, for in its wards the insanities can be demonstrated as they occur in practice, at the period when the practitioner has to deal with them. Clinical instruction in an asylum is all very well in its way, but it is not worth argument to show the infinitely greater advantage that would accrue to all students were such wards open to him.

Let us look to what is done abroad. To each one of the twenty Universities of Germany a psychiatric clinic is attached, under the superintendence of the Professor of Psychiatrie. The buildings are either independent, in the neighbourhood of a general hospital, or they are separate blocks of a hospital, or they are wards specially devoted to the purpose. In many instances they have been in existence for long; the clinic at Heidelberg, for instance, was established in 1878. They vary in size; Würzburg has 60 beds; Tübingen, perhaps the most perfect institution of the kind, holds 120 beds. All alleged to be mentally unsound pass through these hospitals; if the nature of the case demands certification it is passed on to the asylum; the rest, consisting of early or mild cases of insanity, neurasthenia, the subjects of delirium due to fever or consequent on such diseases as pneumonia, post-operation cases, or neurasthenics—in fact, all members of the class which demands observation and treatment are retained without certification and treated to a termination without being reported to the State Office. These clinics are exactly on the same footing as the other clinics, medical and surgical, existing in all German Universities. If not to such a full extent as in Germany, similar provision is made in Austria, Italy, Switzerland, and the United States. Serieux puts it well when he says: "The beneficial influence exercised by the twenty University Clinics of Germany is easy to realise. They have given a vigorous impulse to the study of mental diseases; they form the scientific centres or nurseries for the proper education of the medical profession in psychiatry, and they present an ideal at which the provincial asylums should aim, viz., to become hospitals for the treatment of diseases of the brain."

I venture to think that my main contention is proved by the facts adduced. In most European countries and in Scotland the treatment of recent and acute insanity can be conducted in accordance with law under conditions best suited to procure rapid recovery. In England such is not the case. As regards the treatment of patients derived from the richer sections of the community, the practitioner has to sail very near to the wind of the law. I confess that on several occasions when treating cases south of the border I have deliberately broken the law, and, should occasion arise, I shall repeat the offence in the interest of my patient. We in England and Wales are hampered by a law which prevents us in many instances from doing our duty to the opulent victim of insanity.

From a numerical point of view this is of comparatively small importance. The larger question applies to the accumulation of pauper lunatics in asylums, consequent on their not being submitted to treatment during the period in which morbid conditions are readily amenable to curative influences. If we are to learn anything from the experiment conducted in Glasgow, it is to the effect that it is highly desirable that all cases reported to the authorities as presenting mental symptoms should pass through the sieve of such an institution as there exists. The medical officer in charge can discriminate during a longer or shorter period of observation between those requiring transmission to an asylum, those who can be relegated to a general hospital, those who can be boarded out in private dwellings, and those who can be treated in the special hospital.

The public must be brought to see that the medical is closely connected with the fiscal position. At present it believes that it has done its duty in providing treatment in asylums, although it sorely resents the constantly increasing expenditure. If we can force in on the public mind that early treatment means economy we will soon gain sympathy and support. In all probability an amending Bill will be introduced this session, and, if the members of this Society, independently and collectively, make their views known to the Legislature, the first step towards reform will have been taken. There exists a substantial number of members of Parliament who would support the demand for a Commission to enquire into the whole subject, and not a few who would generally interest themselves in the general question. But the application must be supported by the leading members of the medical profession, by the leading scientific societies, and, if possible, by the Royal Colleges of Physicians and Surgeons. In the event of a Commission not being acceded to by the Government, on the introduction of an amending Bill, pressure should be applied in order to obtain provisions:—

(*a*) For the increase of the medical Commissioners.

ON THE INTRINSIC FIBRES OF THE CEREBELLUM, ITS NUCLEI AND ITS EFFERENT TRACTS.

BY R. H. CLARKE, M.B.

AND

SIR VICTOR HORSLEY, F.R.S., F.R.C.S.

From the Laboratory of Chemical Pathology, University College London.

INDEX.

(1) General Introduction.
(2) Historical Introduction.
(3) Anatomical Introduction.
(4) Results.
(5) Summary.
  (*a*) Relation of the cortex cerebelli to the cerebellar peduncles.
  (*b*) Relation of the cortex cerebelli to the nuclei cerebelli.
  (*c*) The arcuate system.
  (*d*) Size of the intrinsic fibres of the cerebellum.

(1) GENERAL INTRODUCTION.

WE desire at the outset to express our acknowledgments to Dr. T. Woulfe Flanagan for much kind help in the preparation and execution of this work.

The cerebellum, both in its structure and function, has been the subject of much valuable research during recent years at home and abroad.

The anatomy of its lobes and main tracts, their relationship to each other, and the effect upon function of the removal or injury of each of them, have been established on a sound basis.

Further progress must largely depend upon a fuller knowledge of its minute structure; and the series of experiments set forth in this paper were undertaken to obtain more precise data on the true anatomical relations of the cortex of the cerebellum with its own and neighbouring nuclei, and on the actual origin of the efferent tracts.

Two other questions were kept specially in view :—

(1) Is there a direct efferent tract from the cerebellum to the spinal cord ?

(2) And if such a tract really exists, is it derived from the cortex or from the nuclei ; and, in the latter case, from which nuclei ? (*cf.* Thomas).

Until the introduction of Marchi's method the solution of all these questions was impossible, as neither the Weigert-Pal, nor any other method, is sufficiently delicate to distinguish the degeneration of individual fibres, or of small tracts. However, since Marchi's method came into general use relatively few experiments have been directed to the differentiation of the cerebellar cortex from its neighbouring nuclei, and of these none have been carried out with sufficiently minute precautions to make the results absolutely definite. Thus in Thomas's admirable work *Le Cervelet*, records of forty-two degeneration experiments on the cerebellum are given. The animals died in nearly half the cases, and in eighteen they lived less than ten days, while in a large number adventitious lesions were noted. Similarly, in Probst's valuable paper, in no case apparently was the lesion strictly confined to the cortex cerebelli. It is very difficult and sometimes impossible to avoid all accidental injuries, especially such as are due to slight bruising or strain of the peduncles, but the results obtained in such cases ought to be accepted with reserve, and only admitted in corroboration of those where no such mischances have occurred. We would also suggest that when an animal succumbs within a shorter period than three weeks of the operation, the absence of visible degeneration should not be accepted as definite proof that none exists ; since for adequate demonstration of degeneration three weeks is approximately requisite in the majority of animals.

In animals which have not survived more than a fortnight after an operation it is common to find that regions, where extensive degeneration must certainly exist, show very little or none at all. The negative evidence depending on the complete absence of stained fibres in a particular region is conclusive in a well-stained specimen, but is worthless in the case of an animal which has not survived a lesion more than a fortnight. And, again, not only suppuration but any grave constitutional symptoms which may be connected with inflammatory changes near the lesion should, we think, render conclusions drawn from the presence of degenerate fibres of little value, unless these conclusions are very clearly corroborated by other evidence.

We have endeavoured to avoid such sources of error as far as we were able. The animals whose cases we have accepted as available for this paper were all kept three weeks before they were killed under chloroform; they were well nourished and in good health; the operation wounds all healed at once, and required no second dressing. Chloroform was employed for the operations, which were performed by one of us (V. H.), and the animals were always kept fully anæsthetised. The central nervous system was examined after fixing in Müller's fluid and staining in Busch's modification of Marchi's fluid, by being cut in serial sections. By our experiments we have accumulated sufficient data to justify a positive answer to the questions proposed.

From twenty-three uncomplicated lesions (in seventeen cats, two dogs, four monkeys, Macacus Rhesus) of the lateral lobes, and of the anterior and posterior vermis the nuclei being absolutely uninjured in thirteen cases, we have obtained well-marked and well-stained degenerations.

All the specimens show abundant arcuate fibres connecting various parts of the cortex, and other fibres passing to the nearest cerebellar nuclei, whether roof, globose, or dentate, but none going directly from the cortex cerebelli to any of the peduncles. Our series of sections also show that all efferent cerebellar cortical fibres in the first instance pass to one or other of the central nuclei, and principally to the one which is nearest to their origin.

(2) HISTORICAL INTRODUCTION.

The literature of research on the cerebellum, both anatomical and physiological, is already voluminous.

Yet hitherto but few definite facts have been obtained on the special object of our investigation. The reasons for this have been already fully explained.

The following is a list, chronologically arranged, of the chief authorities on the cerebellum who have preceded us, with a brief summary of their results on the special points referred to.

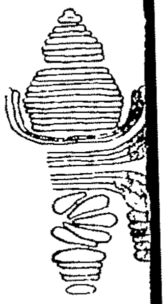
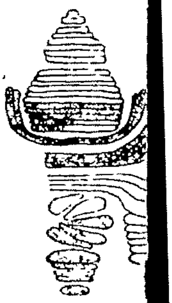
(I.) *Marchi* (1891-1892), to whom belongs the merit of devising the valuable method by which many observers, including ourselves, have obtained their results, was led at the beginning of the investigation of this complex subject to take the view that lesions of the cerebellum (*i.e.*, cortex and nuclei together) caused degeneration of descending tracts from the cerebellum to the spinal cord. Subsequent research has proved that such descending degenerations were the consequence, not of lesions of the cerebellum, but of parts of the mesencephalon and bulb.

(II.) *Ferrier* and *Turner* (1894).—These authors employed the Weigert-Pal method of staining, and were therefore precluded from observing the precise connection between the cortex and the nuclei of the cerebellum. They arrived, however, at the conclusion that injury of Deiter's nucleus was necessary to evoke any degeneration of descending tracts in the spinal cord.

(III.) *Risien Russell* (1894-1896).—The object of Dr. Russell's researches was the determination of the relations between the cerebellum as a whole and the rest of the cerebro-spinal axis. To this end the lesions were designedly extensive, and, consequently, involved the nuclei of the cerebellum. He employed the Marchi method, and established on an irrefragable basis the conclusion that no direct paths exist between the cerebellum and the spinal cord.

(IV.) *Biedl* (1895).—The degenerations observed by Biedl in the spinal cord in his experiments were certainly due to the influence of the lesion on neighbouring portions of the bulb, and, therefore, although his work appeared to support the original idea of direct connection between the cerebellum and the spinal cord, he does not regard it now as in fact doing so.

Arcuate Fibres.	Remarks.
w fibres ascend to *pyramid* and lia of *para-median lobe.* A few to *culmen* and *lobus centralis.* o opposite nucleus, fastigii and of folia, posterior to primary e fibres pass down to the *para-* res thus passing to the principal ermis cross the mid line.	
... ...	Fine fibres pass across the medulla of the *vermis*, but none in front of the commissure. A few fibres pass to the lateral region of *corona*, *i.e.*, to the folia of the pennate gyri.
y few fine fibres to folium im- jacent to slightly bruised folia.	The fibres described as entering the *nucleus fastigii* run straight inwards along the roof of the *recess* of *Retzius*.

No.	Lesion.	D Shaded area indic explanat
Cat 4 ...	Bruising and laceration of surface of all folia of *lateral lobe*, excepting the posterior pennate and posterior paraflocculur lobules.	
	Combined Lateral Lobe and Vermis Lesion. (5)	
Cat 5 ...	Two parts:— I.—*An upper horizontal incision* which separates (*a*) *on left side* the cortex of five folia of the *culmen*, and also of the lateral lobe. On (*b*) the *right side* the lesion extends horizontally to the same distance, *i.e.*, to the dorsal paraflocculus, but sparing in part the two most caudal folia of culmen. II.—*An oblique incision* through the pyramid into the fourth venticle, completely detaching the uvula, nodulus, and ventral half of the pyramid.	 *Note*—Lesion of sponds See photog
	Vermis (anterior) Lesions, specially including the Culmen. (6) *Culmen.* (7) *Culmen and all the middle lobe.* (8) *Culmen : lobus centralis and first two lobules of middle lobe.*	
Rhesus 6 ...	The lesion consists of a horizontal puncture entering the culmen close to the mesial plane just in front of the primary fissure, undermining the five posterior folia of the culmen. The puncture in its greatest depth reaches just to the dorsal limit of the central white mass of the lobe.	See photograph,

No.	LESION.	DIAGRAM. Shaded area indicates les explanation see
Rhesus 22 ...	(1) Superficial removal of uvula and bruising of pyramid, and slight bruising of posterior folium of nodulus. (2) Deep destruction of uvula fibres and of nucleus fastigii; bruising of nucleus globosus on right side. [Nucleus globosus and fastigii on left side normal.] Bruise of nucleus cuneatus, right side.	See photograph, Plat

No.	LESION.	DIAGRAM. Shaded area indicates les explanation see
Rhesus 23 ...	(1) Eight folia of lobus quadrangularis, mesial third. (2) Neighbouring border of corresponding eight most posterior folia of the culmen.	See photograph, Plate

(V.) *Münzer* and *Wiener* (1895).—These authors also found no fibres proceeding directly from the cerebellum to the spinal cord.

(VI.) *Thomas* (1897).—The most extensive and thorough investigations on the present subject have been undertaken by Thomas. The general results he obtained may be summarised as follows:—

(1) Removal of the cortex of one cerebellar hemisphere produces no degeneration of the fibres in the spinal cord.

(2) Degeneration in the spinal cord is directly proportionate to injury of the vestibular nuclei, *i.e.*, Deiters' and Bechterew's.

(3) The cerebellar nuclei, *i.e.*, nucleus dentatus and nucleus fastigii (vel tecti), send fibres to the vestibular nuclei.

(4) The cortex of the hemispheres (*sic.*, *query* lateral lobes) appears to be the principal source of fibres passing from the cerebellum to the pons. Of all the fibres in the middle peduncle, however, but few relatively are cerebello-pontine; the majority are ponto-cerebellar.

(5) The cortex is directly connected with the nucleus tecti and the nucleus dentatus.

With all these conclusions our own results are in complete accord. But Thomas is further of opinion that some fibres leave the cerebellar cortex and pass by means of the restiform body to the nucleus lateralis and nucleus cuneatus of the bulb. He also leaves it undecided whether fibres do pass from the cortex to the superior cerebellar peduncle. The first of these points is contradicted by our experiments, and the second we are able to decide in the negative.

(VII.) *Ramon y Cajal* (1901).—This investigator has suggested that some fibres may pass directly from the cortex to the superior peduncle. His definite conclusions, as given in his text-book, however, are as follows:—

(1) The axones of the Purkinje corpuscles of the cortex terminate in the nucleus dentatus and the nucleus tecti.

(2) The axones of some of the cells of the nucleus tecti pass down the spinal cord to come into connection with the cells of the anterior cornua.

(3) The axones of the cells from the dentate nucleus bifurcate, one limb entering the superior peduncle, the other passing down the lateral region of the bulb and spinal cord, but with indefinite termination.

In other parts of his work he apparently accepts the position put forward by Marchi, and it is to be observed that he always specially speaks of the descending spinal cord fibres as the "*via descendente de Marchi.*"

He also gives a figure of descending degeneration in the spinal cord of a guinea-pig after extirpation of one cerebellar hemisphere, but it is obvious from the other facts of the experiment that the lesion was a very extensive one.

(VIII.) *Probst* (1902).—Since the work of the investigators hitherto cited a systematic research on the afferent and efferent tracts of the cerebellum has been published by Probst. His plan was the same as ours, namely, to make lesions with aseptic precautions relying entirely on the Marchi method to follow the degenerated fibres. He does not anywhere describe that the lesions were absolutely restricted to the cortex of the cerebellum, and, in fact, all seem to have been combined with lesions of the cerebellar nuclei in varying degree of intensity. Although in our opinion it is not justifiable to argue that inasmuch as the number of fibres issuing in the cerebellar efferent paths is apparently proportional to the amount of the injury that the nuclei sustains, and therefore that no fibres pass from the cortex to the peduncles directly Probst considered that he was able to formulate the following conclusions:—

(1) That no fibres go from the cerebellar cortex into the spinal cord or into the thalamus.

(2) All the centrifugal fibres of the cerebellar cortex pass to the dentate nucleus or the roof nucleus or to Deiter's nucleus.

(3) Direct fibres from the cerebellar cortex can be traced to the pontine nucleus.

(4) Direct fibres pass from the cortex in the outer segment of the restiform body to the nucleus of the lateral columns.

(3) ANATOMICAL INTRODUCTION.

(1) *Cerebellum of the Cat.*

The nomenclature of the different parts of the cortex of the cat's cerebellum is unfortunately at the present time under revision.[1] We therefore offer the following description based in the main upon the work of Elliot Smith. While we think it undesirable to employ terms which convey homologies that have not yet been accurately determined, certain names in common use will be found convenient. Thus, the cerebellum for physiological, and in part also for anatomical, reasons, has been divided into a mesial portion or *vermis*, and, on either side of this, a *lateral lobe*, which on the posterior surface of the organ is separated from the vermis by the *sulcus paramedianus*. These names we intend to retain.

The vermis.—This may best be divided, as Elliot Smith proposes, into three lobes—*anterior*, *middle*, and *posterior* (see fig. 1). The delimiting sulcus between the first two is now generally termed the *primary fissure*. This is a deep vertical cleft which is well seen in a mesial section of the whole organ. The cortex in front of this fissure may be called the *anterior lobe*, provided it is fully understood that it is separated on each side from the *lateral lobe* by the *primary fissure* turning downwards and forwards until it arrives at the surface of the *superior peduncle*.

(I.) The *anterior lobe* consists of some twenty-one folia which have been divided from below upwards into: (1) The *lingula*, consisting of one, or at the utmost three, folia. (2) The *lobus centralis*. This is composed of about six folia, and extends to the upper surface of the *inter-collicular commissure*. (3) The remainder of the *anterior lobe* is made up of about twelve folia, and has usually been described as the *culmen*, by which term we shall speak of it.

For the sake of simplicity we will next dispose of :—

(II.) The *posterior lobe*, which is formed by only two lobules, namely, the *nodulus* and the *uvula*, and is limited

---

[1] This is not the place to refer to the very important works on this subject by Bolk, Charnock Bradley and Elliot Smith ; we can only refer the reader to their papers.

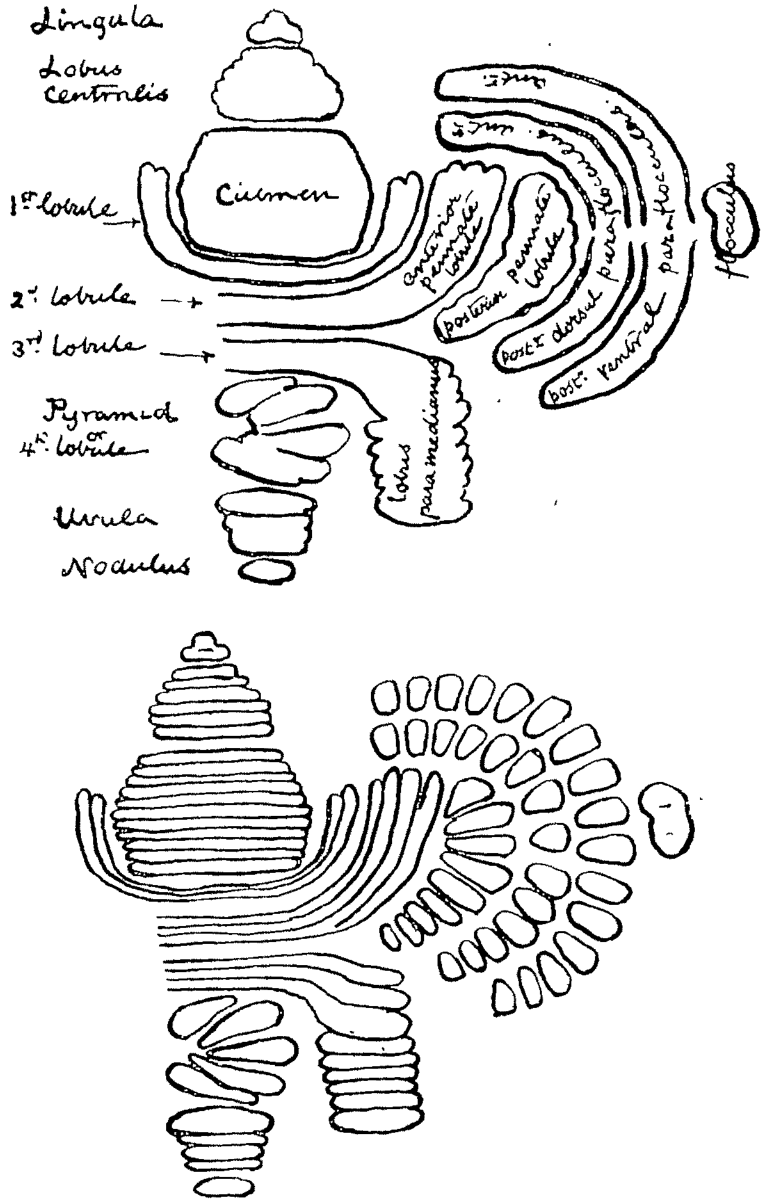
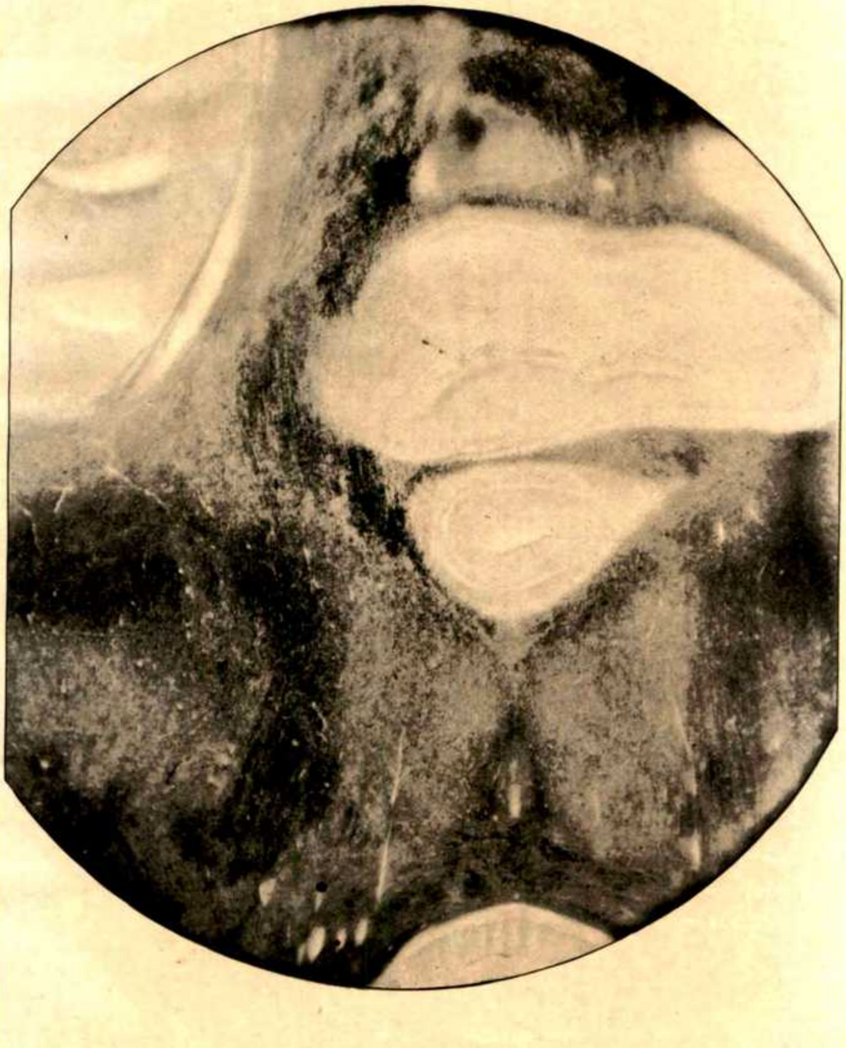
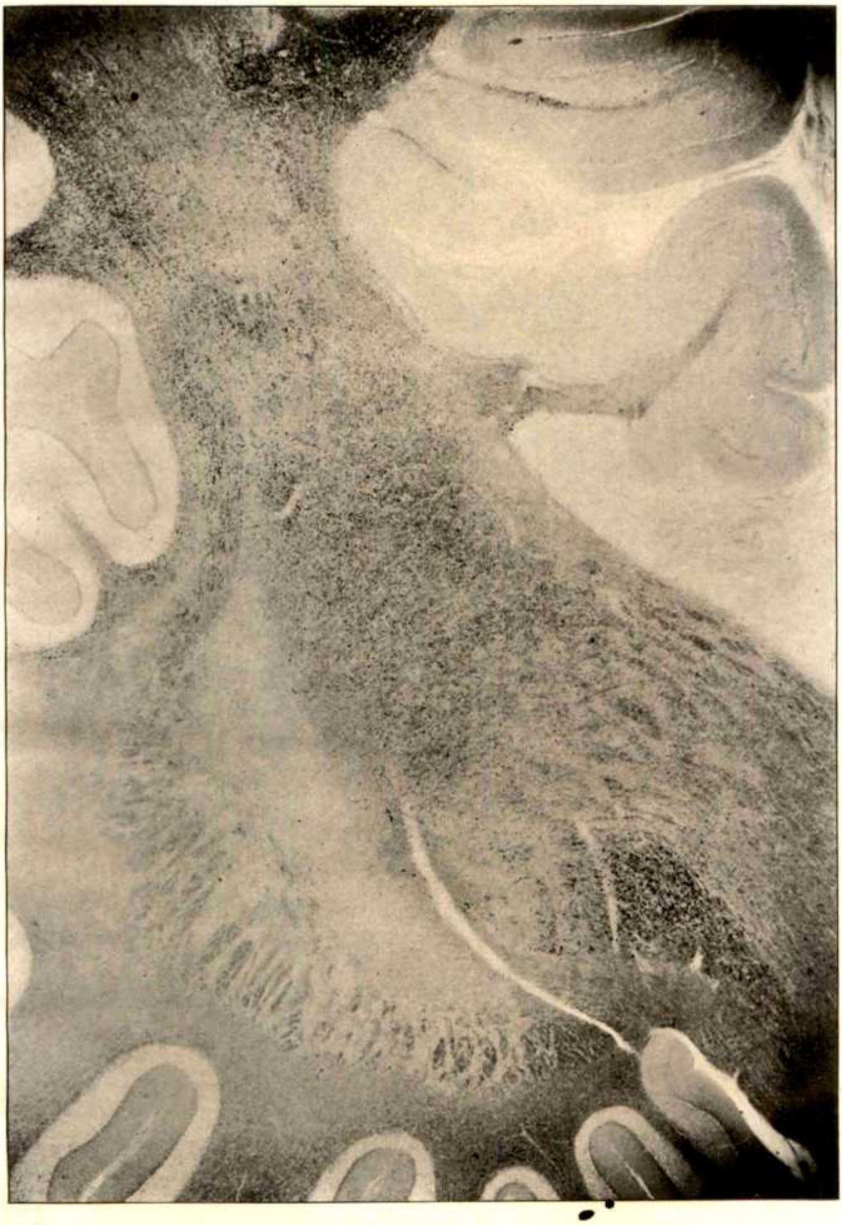


FIG. 1.

Cerebellum of cat. The cortex of the vermis and lateral lobes represented on the flat.





dorsally by a deep sulcus, the *secondary fissure* of Elliot Smith. The *nodulus* consists of only one folium which often has a double head. The *uvula* contains from four to five folia, the most dorsal of which lies on the *obex* of the *calamus scriptorius*, is always a large folium, and is bounded on its dorsal surface by the *secondary fissure.*

(III.) The *middle lobe* has been divided by Elliot Smith into four parts.[1] The (*a*) *first lobule* consists on the average of two folia which, situated like a horseshoe behind the anterior lobe, form the posterior lip of the primary fissure. The (*b*) *second lobule* is composed in the middle line of two or three folia, which spread out laterally into a series of folia arranged bi-pennately on the supero-anterior aspect of the *lateral lobe.* The anterior series of these pennate folia are usually from four to five in number, their long axes are antero-posterior, and they form a very definite quadrangular topographical feature of the roof of the *lateral lobe.* We shall speak of them collectively as the *anterior pennate lobule.* From the outermost of these five antero-posterior folia a series of about eight folia form the tip of the *lateral lobe,* and are continued posteriorly behind the antero-posterior folia before mentioned, so as to complete, though somewhat asymmetrically, the bi-pennate arrangement of the roof of the *lateral lobe.* They may be termed the *posterior pennate lobule.* Inasmuch as the side of the *lateral lobe* on its outer aspect is built up, as will be seen directly, of the *floccular lobes,* the bi-pennate arrangement of the roof together with the *paraflocculus,* has not inappropriately been termed by Elliot Smith the *suprafloccular mass.* We have above suggested that this bi-pennate roof of the lateral lobe should be divided into a *pars anterior* (the anterior pennate lobule) and a *pars posterior* (the posterior pennate lobule). (*c*) The *third lobule,* which forms in the middle line the upper horizontal limb of the convoluted posterior *vermis,* includes from two to three folia, and extends outwards on each side into two vertical lobes forming the outer boundary of the *paramedian fissure,* and, therefore,

---

[1] Some French writers speak of the whole *vermis* as the middle lobe of the cerebellum.

P16253

properly termed the *paramedian lobes* ; each of these is made up of from nine to ten folia. In contact with the outer surface of this vertical lobe lies the posterior border of the *parafloccular mass*. (*d*) The *fourth lobule* is formed by the *pyramid*, and, in the cat, includes from four to five folia placed obliquely since it enters into the sigmoid convolution of the posterior vermis forming its lower horizontal branch.

We will now describe the remainder of the *lateral lobe* (or hemisphere of human anatomy), *i.e.*, the ventral two-thirds of the depth of the anterior, external and posterior aspects of the outer part of the lobe.

On the external or lateral aspect the folia are apparently arranged in three tiers, but on the anterior and posterior surfaces in only two. The lowest tier on the external aspect is made up anteriorly of the sessile *flocculus* and posteriorly by the *tuberculum acusticum*. The remaining two tiers extend horizontally round the lobe both forwards and backward, and are termed the *dorsal* and *ventral paraflocculus*. A portion of the *ventral paraflocculus*, where the anterior and exterior surfaces meet, is embedded in a special fossa of the *petrous bone*, and is spoken of as the *petrosal lobule*. Each tier of the *paraflocculus* is composed of from fourteen to sixteen folia.

These terms by which the various areas of cortex thus marked off may conveniently be known must, in the present state of our knowledge, be merely provisional until the exact homologies are ascertained. We therefore furnish diagrams of the lesion in each experiment, and photographs where these have been specially taken. The precise area of the lesion, however, is seldom well shown in a photograph of the actual specimen as when the dura mater is adherent to the neighbourhood of the wound the surface is necessarily ragged after dissection, and, on the other hand, undermined folia often appear normal (*cf.* plate IV.).

(4) RESULTS.

We have thought it better to group our results in tabular form (Table I.). The columns of the table sufficiently explain themselves, but it will be understood that where the definite statement "*nil*" is made it means that there were no degenerated fibres to be found in that part after repeated re-examination of the various series of sections. It will be seen that there is a very close concordance of results obtained from lesions of the same district of the cerebellar cortex. We think that the explanation of this is to be found in the very simple plan on which the cerebellum is constructed and the homogeneity of its structure throughout.

(5) SUMMARY.

(*a*) *Relation of the Cortex Cerebelli to the Cerebellar Peduncles.*

We believe that the results of our experiments embodied in the foregoing table justify the following general conclusions on the direct question whether fibres (presumably axones of the Purkinje corpuscles) pass directly into the cerebellar peduncles or not.

(1) No fibres issuing from the cortex cerebelli enter any of the peduncles.

(2) All fibres leaving the cerebellum by way of the peduncles have origin in one or other of the cerebellar nuclei.

(*b*) *Relation of the Cortex Cerebelli to the Nuclei Cerebelli.*

Our observations show that any given area of the cortex is in relation with a homolateral nucleus or nuclei. We have not found satisfactory evidence that the cortico-nuclear fibres cross the mesial plane.

This simple relation of the cortex and nuclei is illustrated in fig. 2, where, from a lesion in the vermis, fine fibres are seen streaming forwards to reach the dorsum of the roof nucleus.

The details of these relations are perhaps best shown in parallel columns as follows:—

TABLE II.

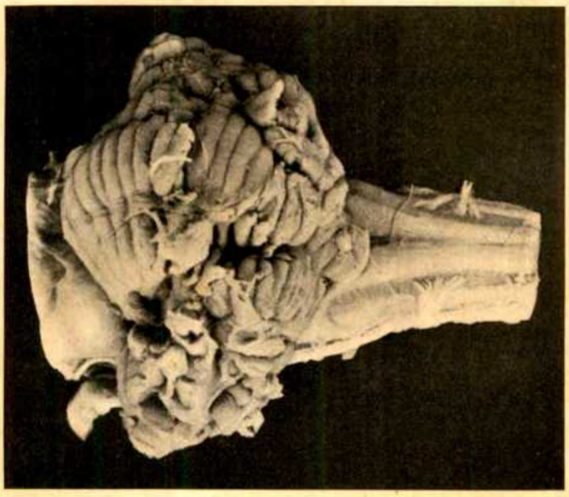
Portion of Cortex Cerebelli.	*Nucleus with which it is in relation.*[1]
(1) Anterior pennate lobule.	Nucleus fastigii. Nucleus dentatus.
(2) Posterior pennate lobule.	Nucleus dentatus.
(3) Flocculus, paraflocculus.	Nucleus fastigii.
(4) Anterior pennate lobule. Anterior Paraflocular lobule. Paramedian lobe.	Nucleus dentatus. Nucleus globosus. Nucleus fastigii. Nucleus vestibuli.[2]
(5) Omitted for involvement of nucleus fastigii.	
(6) Culmen (five posterior folia).	Nucleus fastigii. Nucleus globosus.
(7) Culmen and all middle lobe.	Nucleus fastigii. Nucleus dentatus. Nucleus vestibuli.[2] Nucleus globosus.
(8) Left half of lobus centralis, culmen and left four-fifths of first and second lobule of middle lobe.	Nucleus dentatus. Nucleus fastigii. Nucleus globosus. Nucleus vestibuli.[2]
(9) Second lobule of middle lobe and anterior pennate lobule.	Nucleus dentatus. Nucleus fastigii. Nucleus globosus. Nucleus vestibuli.[2]
(10) Three folia of third lobule of middle lobe	Nucleus fastigii (very few fibres).
(11) Third and fourth lobules of middle lobe.	Nucleus fastigii. Nucleus globosus. Nucleus dentatus.
(12) Fourth lobule of middle lobe (pyramid).	Nucleus fastigii.
(13) Fourth lobule of middle lobe and two outer folia of uvula.	Nucleus fastigii.
(14) Lowest folium of fourth lobule of middle lobe and two folia of uvula.	Nucleus fastigii.
(15) Whole uvula.	Nucleus fastigii. Nucleus globosus.
(16) Ventral half of fourth lobule of middle lobe, uvula, and two anterior folia of first lobule of middle lobe.	Nucleus fastigii. Nucleus globosus.
(23) Lobus quadrangularis and side of culmen.	Nucleus fastigii. Nucleus globosus. Nucleus dentatus.

---

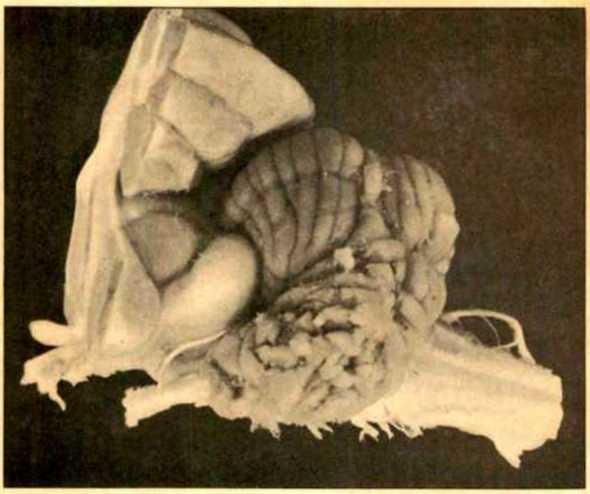
[1] Where more than one is concerned the nuclei are arranged in the order of more extensive anatomical connection.

[2] It will be observed that the association of the nuclei vestibuli has only been noted in extensive lesions. It is conceivable that secondary circulatory changes may possibly account for the more extensive association.

PLATE I.



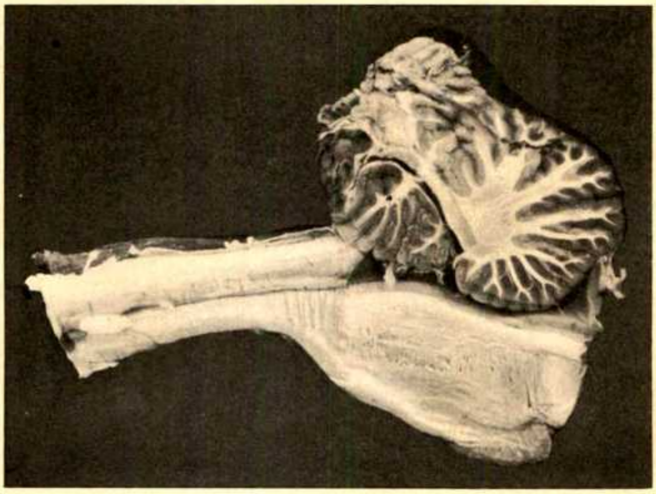
Experiment No. 1.



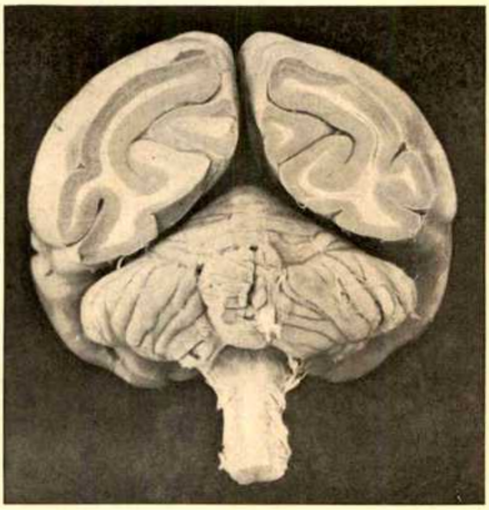
Experiment No. 2.

The numbers refer to the Experiments given in Table I.

PLATE II.



EXPERIMENT No. 5.



EXPERIMENT No. 6.

The numbers refer to the Experiments given in Table I.

From the foregoing it is evident that the *roof nucleus*, nucleus fastigii (see fig. 2), is of chief importance among the nuclei, for it is in relation practically with all parts of the cerebellar cortex, and must consequently possess a very important function in associating the cortico-fugal impulses.

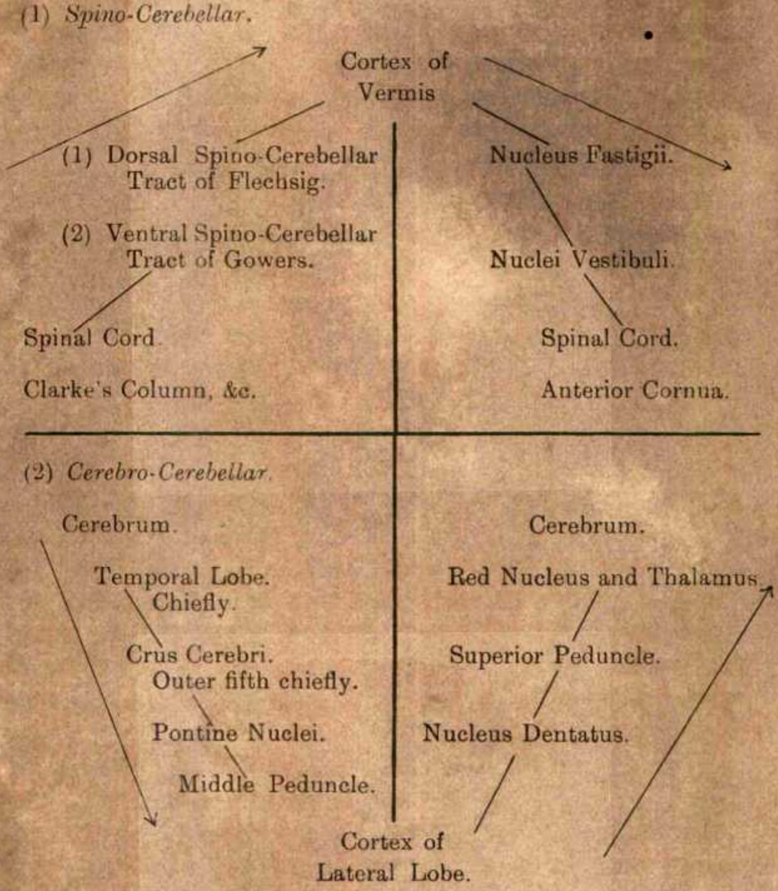
Further, it has for long been known to have special association with the vestibular nuclei (Kölliker, Edinger, Ramon y Cajal). In our preparations, where very limited lesions of the nucleus fastigii had been made, it has been quite easy to follow both coarse and medium fibres over the superior cerebellar peduncle to the gray matter surrounding that structure, and to the locus cœruleus (see fig. 3). We would suggest the terms "fastigio-brachial" and "fastigio-cœrulean" to denote these fibres, but their full discussion and description must be reserved for another communication.

The *nucleus globosus* has no relation with the lateral lobe or flocculus. It is essentially a nucleus appertaining to the vermis system, and its connections extend as far laterally as the paramedian lobe.

The *nucleus dentatus* is intimately associated with the pennate lobules, but not with the flocculus and paraflocculus. These two important portions of the cortex are connected with the roof nucleus, which fact fully confirms the anatomical conclusions of Elliot Smith as regards the relation of the floccular region to the postero-inferior part of the vermis.

As regards any association of the nucleus dentatus with the mesial mass of the cortex or vermis, our experiments suggest clearly that, first, there is no relationship between the nucleus dentatus and the anterior or posterior lobes, *i.e.*, lingula, lobus centralis, culmen, uvula, and nodulus; and, secondly, that the only connection between the nucleus dentatus and the vermis is with the middle lobe of the latter. Finally, of the middle lobe our evidence goes to show that it is only the three anterior lobules which are connected with the dentate nucleus, and that the fibres making such connection are relatively few.

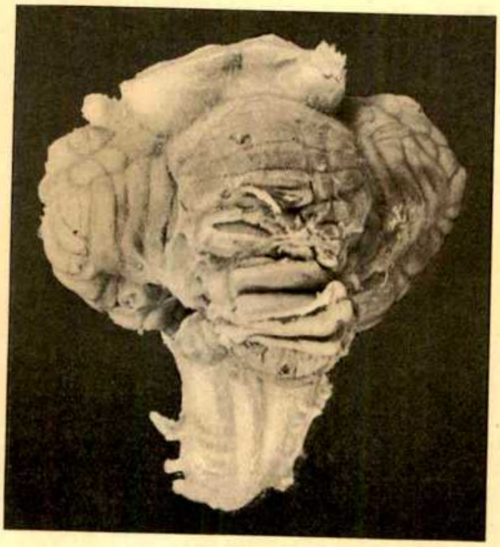
The intrinsic arrangement of the cerebellar cortex and nuclei can be therefore regarded as divisible into two distinct organs, a spino-cerebellar and a cerebro-cerebellar looped system, of which the parts are thus arranged :—



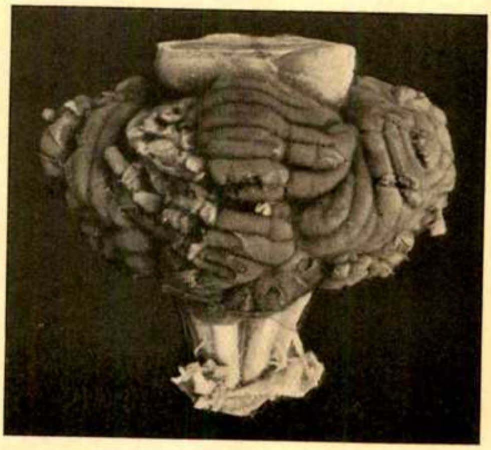
In considering the relation of the cerebellum to the cerebral pallium, much stress has been laid in the past on the excitable or so-called motor area of the cortex cerebri as being probably that part of the pallium which is definitely associated with the pontine nuclei and so with the cerebellum.

That this association exists is amply established by the investigations of Luciani, Ramon y Cajal, Simpson,

PLATE III.



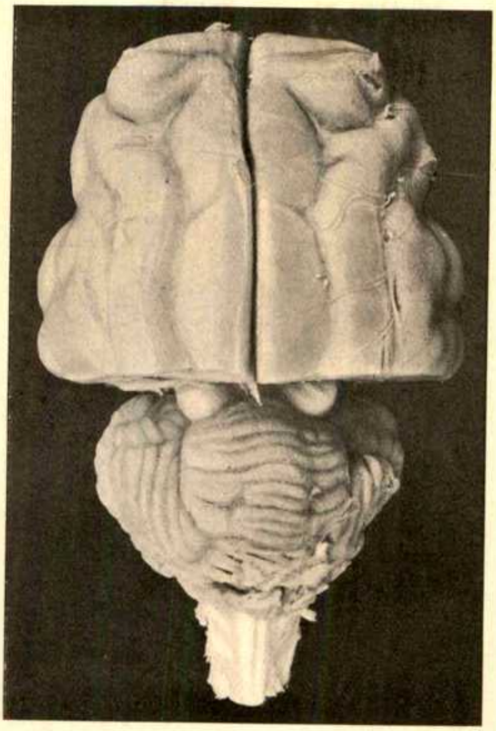
EXPERIMENT NO. 7.



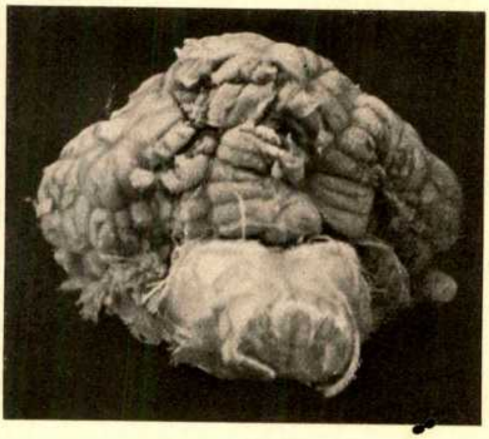
EXPERIMENT NO. 8.

The numbers refer to the Experiments given in Table I.

PLATE IV.



Experiment No. 9.



Experiment No. 10.

The numbers refer to the Experiments given in Table I.

Beevor and Horsley, proving the existence of an extensive system of intrapontine pyramidal collaterals.

It appears to us, however, that this point requires further consideration, and, as we have in the above scheme put the temporal region as being that part of the cortex cerebri which is most directly connected with the cerebellum, we must now state the grounds for this view.

Taking first the crural tracts, the opinion of Dejerine is, of course, universally recognised to be correct, viz., that the temporo-pontine bundle occupying the outer fifth of the cerebral peduncle finds origin in the middle third of the temporal lobe and terminates in the pons (frontal segment according to unpublished experiments by Beevor and Horsley). Now, inasmuch as the temporal pallium is the seat of projection of the auditory paths, it is not unreasonable to regard it, rather than the excito-motor gyri, as being also the area in which the vestibular sensations are represented.

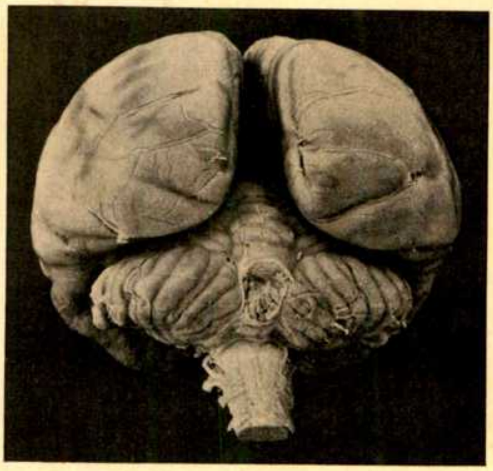
This view of the pallial vestibular representation has also received the authoritative support of Chas. K. Mills, who, in his most recent diagram (*Medical News*, November 5, 1904), figures "equilibratory" and "orientation" in the posterior third of the temporal lobe (second and third gyri).

Since the ponto-cerebellar fibres cross the raphe, it follows that the temporal pallium of one side would correspond with the opposite lateral lobe of the cerebellum, which is well known to be the case, as far, at any rate, as the whole cerebral hemisphere is concerned.

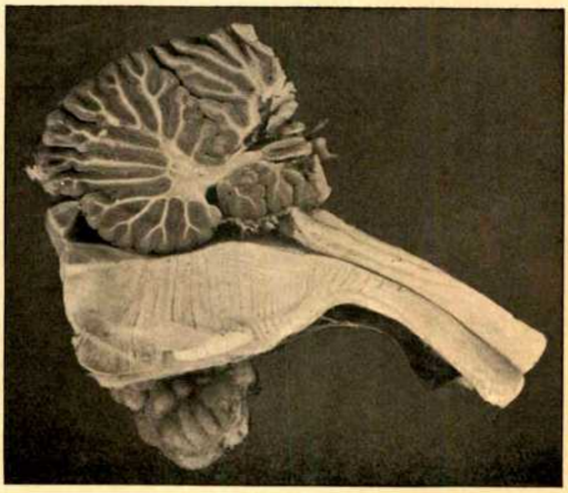
Further, the records of cases of defective development of the cerebellum hitherto published (*cf.* Risien Russell, Herringham, Lewis, &c.) show that the part of the cortex cerebelli to fail first is the lateral lobe, or, in other words, that part which, having cerebro-pallial relations, is necessarily later in evolution and succumbs, therefore, more readily to adverse circumstances.

The solution of this question will afford the basis of a rational explanation of the phenomenon of cerebellar circumduction, or "*mouvement de manège*," but at the present time it is uncertain to what degree "*mouvement de manège*"

PLATE V.



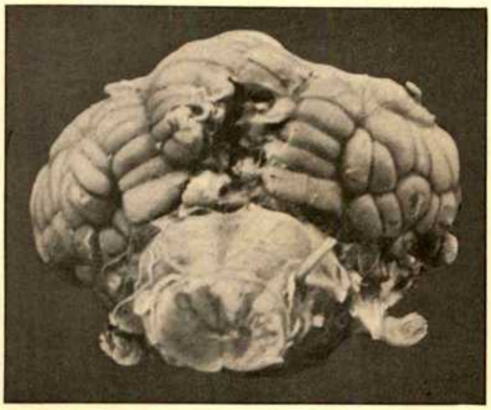
Experiment No. 12.



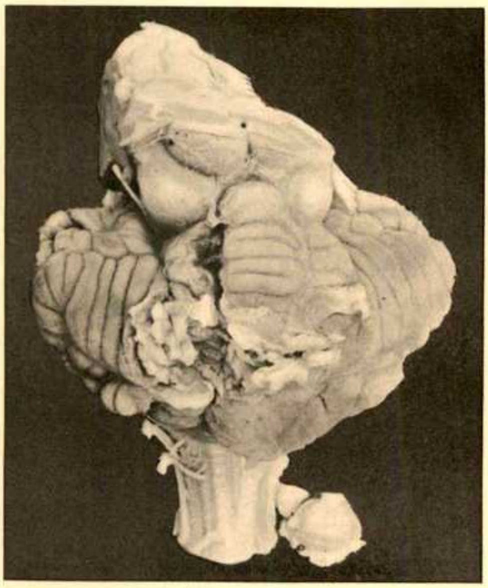
Experiment No. 16.

The numbers refer to the Experiments given in Table I.

PLATE VI.



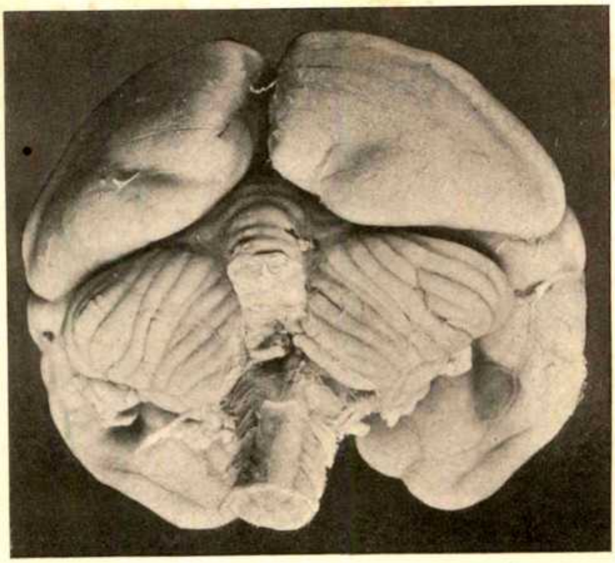
EXPERIMENT No. 17.



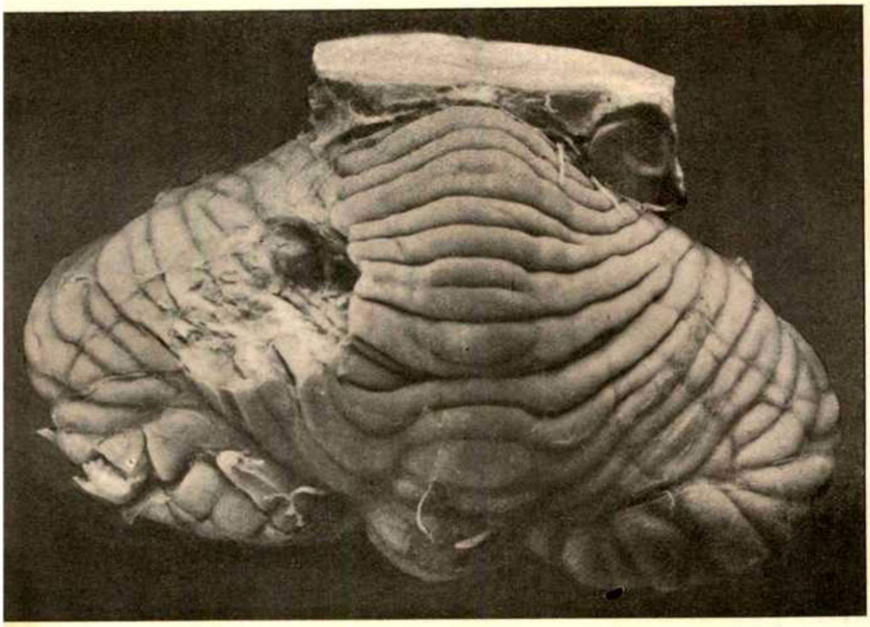
EXPERIMENT No. 18.

The numbers refer to the Experiments given in Table I.

PLATE VII.



Experiment No. 22.



Experiment No. 23.

The numbers refer to the Experiments given in Table I.

(3) Nucleo-peduncular: Superior, large; middle, fine; inferior, medium.

The differences in size of the various groups are well shown in fig. 3, which is a photograph of a section showing the cortico-nuclear and nucleo-fugal fibres in their relative proportions in the cat.

REFERENCES (pp. 16-18).

(I.) "Sull' origine e. decorso dei peduncoli cerebellari, &c," *Firenze*, 1891, and *Riv. Sper. d. Fren. s. Med. Leg.*, 1886-1891.
(II.) *Philosophical Transactions of the Royal Society*, 1894.
(III.) *Philosophical Transactions of the Royal Society*, 1896.
(IV.) *Neurologisches Centralblatt*, 1895, p. 434.
(V.) *Prager Medizinische Wochenschrift*, 1895, No. 14.
(VI.) *Le Cervelet*, Paris, 1897.
(VII.) "Textura del Sistema Nervioso, &c." Madrid, 1899-1902.
(VIII.) *Archiv. f. Pschiatrie*, xxxv., p. 692.

DESCRIPTION OF PLATES.

The plates hereto appended are the actual photographs taken of the cerebellum in most of the cases included in Table I.

Reference must be made to Table I. for the accurate delimitation of the lesion especially where this has had an undermining effect.

DIFFUSE SARCOMATOUS INFILTRATION OF THE SPINAL PIA MATER.

BY STANLEY BARNES, M.D., BSc., M.R.C.P.,
Pathologist and Medical Registrar to the Queen's Hospital, Birmingham.

ALTHOUGH many cases of multiple tumours of the central nervous system have been published, yet the means whereby these tumours are spread are still very imperfectly understood. If we are to assume that in the matter of new growths the central nervous system is analogous to the other parts of the body, we are driven to adopt the hypothesis that, as a general rule, a new growth there is primary in only one situation, and that in any cases of multiple growths one of the lesions was primary and the others were secondary—*i.e.*, are the result of metastatic "infection." It therefore becomes a matter of considerable interest to ascertain whether the means of dissemination can be explained adequately in the present state of our knowledge in the various cases now published in the literature.

Two cases recently occurred at the Queen's Hospital, Birmingham, which throw considerable light on this subject, and, in recording the clinical and *post-mortem* phenomena, I have ventured also to append an abstract of certain similar cases to which I have had access. I am indebted to the kindness of Dr. Carter for permission to publish these cases.

Case 1 (Barnes).—On January 3, 1903, a boy, 13 years of age, was admitted to the Queen's Hospital complaining of progressive loss of eyesight for two years.

Family history.—Father alive and well, has never had syphilis. Mother died at 36, of puerperal fever. There are five brothers and one sister, all well; there have been no other children, or miscarriages. There is no suggestion of syphilis in any of the other children.

Previous history.—Always strong and healthy.

Present illness.—Began two years ago with attacks of morning vomiting, having no relation to food, and with progressive loss of sight About the same time he also complained of occasional headaches, which are stated to have been of only moderate severity. He also became generally weak and wasted, but after an illness lasting about six weeks, he began to improve—the sight improved sufficiently to enable him to read large print, the vomiting ceased, and the headache disappeared. Two months ago the symptoms recurred: morning vomiting, further loss of sight and frontal headache being the prominent symptoms.

State on admission.—A pale, thin boy of 13, lay comfortably in bed. Temperature, 97·5; pulse, 96; respirations, 24.

Nervous system.—Intelligence, memory and speech normal. Smell good on both sides.

Vision was reduced to perception of light only, on both sides. The optic discs both showed optic neuritis with early atrophy.

There was doubtful weakness of the left external rectus muscle, but no obvious squint. The pupils were equal and dilated; they did not react to light.

Taste slow, but otherwise normal. Fifth nerve normal. There was some supranuclear paralysis of the right side of the face. Hearing normal on both sides. 9th, 10th, 11th, and 12th nerves normal.

Motor system.—There was no local wasting or paralysis other than in the face. General muscular wasting considerable. Sensation normal throughout. Co-ordination good. Gait natural for a blind boy.

Reflexes.—Organic, normal. Plantars were lively, with flexor response. Knee-jerks were absent. Abdominal and cremasteric equal and well marked. No trophic disturbances.

The other systems, circulatory, respiratory, &c., were examined and found normal.

Progress whilst under observation.—On January 5 he had an attack of cramp affecting both hands simultaneously and lasting about five minutes; the hands were flexed upon the forearm, the fingers being extended; there was no twitching, and consciousness was not lost.

On January 8, a convulsion occurred. It began with a loud scream, and then at once the back was arched and the legs and arms were extended; the wrists were flexed and the fingers extended; he was unconscious for a few seconds. This was followed a few minutes later by a second convulsion; it was of the same type but was followed by clonic spasms of the left side of the face, of the arm and leg. He was drowsy afterwards, and remembered nothing of the attack.

On January 11, it was noted that the respiration was irregular, being somewhat of the Cheyne-Stokes' type. He was evidently very much worse, and in the night moaned continually in his sleep. Next morning he was very sick, complained of feeling ill, and soon lapsed into a semi-comatous state. The toes of the left foot were flexed, and then spasmodic contractions occurred in the left leg for a few minutes; then gradually the arms were extended and the fingers and thumbs twitched. The left side of the face was also convulsed, and then the back arched, the head was thrown back in tonic spasms for a few moments, and then he gradually relaxed and the fit was over. The time occupied by these movements was about fifteen minutes. He gradually became conscious, but remained very dull.

Next day (January 13) he could be roused and replied sensibly to questions, but tended to drop asleep whilst one was talking to him. Breathing was still very irregular.

On January 17 he was more comfortable, and the headache and sickness had passed off. No more convulsions had occurred. Both external recti seemed partly paralysed, and vision was now absent.

On January 21, he began to pass urine and fæces into the bed; he was now wasting considerably.

From this date forwards he went slowly down hill; he became more and more drowsy, having occasional convulsions of no great severity, and of the type already described. By June 1, he was so wasted that despite the most careful nursing bed-sores occurred on the hips and sacrum; there seemed to be no subcutaneous fat left, and the muscles were reduced to about one-third of their original size. There was now no rigidity, and for several weeks all the reflexes (including the plantar) had been absent.

He died after a short convulsion on June 21, 1903. For a week before death, the temperature had not risen above 95·4°. During his stay in hospital, it never rose above 99°.

He was treated with iodide of potassium for a long time; for the headache, potassium bromide was usually given.

Post-mortem Examination.

The autopsy was performed on June 22, twenty-four hours after death.

The body was greatly emaciated, and rigor mortis was general.

The thoracic and abdominal viscera were carefully examined and found normal. There was no sign of growth, nor was anything found suggestive of tuberculosis or of syphilis.

The head was unusually large, especially in the bi-parietal diameter. The skull was of natural thickness. The dura mater was not thickened, and was everywhere free from adhesions to the arachnoid.

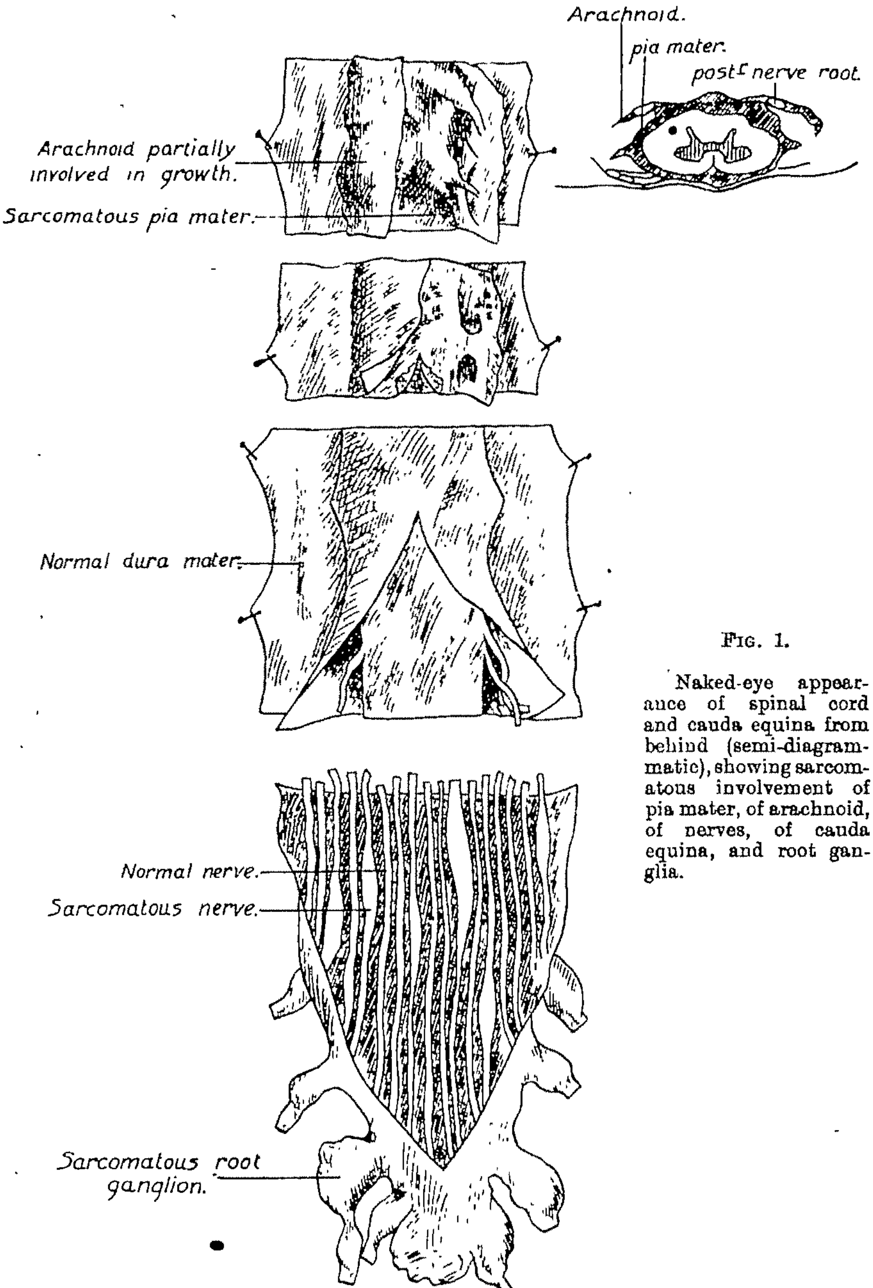
The convolutions of the brain were of natural size and arrangement, and were considerably flattened out. The sulci contained very little fluid.

The pia and arachnoid over the vertex were normal except in the left frontal region; here was a somewhat circular patch about the size of a crown piece, which in the fresh state was of an opaque greenish-white colour, and at first sight appeared to be an island of purulent lepto-meningitis. The arachnoid covering this patch was smooth and not adherent to the dura mater.

At the base of the brain the dura was normal, but the other membranes were involved in a massive infiltration of solid tissue. This thickening of the pia-arachnoid extended as far forwards as the optic chiasma and anterior perforated spaces, and laterally to the apices of the temporo-sphenoidal lobes. All the natural hollows of the base of the brain were more or less completely filled up with this material, and the upper and lower boundaries of the pons were obscured on the surface. The meninges on the under surface of the cerebellum were a little thickened, but at the region where the medulla strikes free from the cerebellum the thickening, though still most marked on the under surface, almost surrounded the spinal axis.

The laminæ were removed and found normal. The theca looked normal externally, and when opened was found to be of natural thickness, and not adherent unnaturally to the subjacent membranes. It was now seen that the cord was almost entirely surrounded by the thickening of the membranes which extended throughout its entire length (see fig. 1). The surface appearance was like that of the material at the base of the brain; of an opaque pinkish colour, it presented many nodosities over its coarsely uneven surface. In the main there was more growth over the posterior than over the anterior regions; the thickness varied up to a maximum of twelve millimetres.

The emerging nerves, cranial and spinal, with the exception of the first pair of cranial nerves, all passed through the thickened



FREDERIC BARKER del.

FIG. 1.

Naked-eye appearance of spinal cord and cauda equina from behind (semi-diagrammatic), showing sarcomatous involvement of pia mater, of arachnoid, of nerves, of cauda equina, and root ganglia.

membranes. The nerve roots were not markedly atrophic. The posterior root ganglia throughout the spinal region were enormously enlarged, those of the cauda equina being about the size of horse-beans; in the cervical region they were somewhat smaller, but the Gasserian ganglia were as large as those of the cauda equina. A few similar discrete nodes were present along the intra-thecal course of the nerves forming the cauda equina.

Many of the peripheral nerves were examined, but nowhere outside the intra-spinal or -cranial course could any thickening be discovered.

A series of horizontal sections was then made through the hardened brain. At the level of the internal capsule, it was found that a large tumour was present in the left frontal region. The tumour as a whole was reddish-grey in colour, with some streaks of yellow near its centre and hinder end; it was about the size of a Tangerine orange, and roughly triangular in shape, the apex being directed backwards and inwards and lying near the foramen of Monroe. The hinder and inner end of this tumour had broken down, clearly rupturing into the lateral ventricle; at this point the tumour was hollowed out and had a rough and uneven base facing the ventricle, and the deeper yellow colour showed that some time previously an extravasation of blood had occurred here.

The growth extended forwards in the white matter from this point as far as the gray matter of the frontal cortex; in one place the tumour had grown along the course of the entering cortical vessels, and then involving the subpial tissues had formed the greenish-grey mass on the surface which had previously been mistaken for inflammatory exudate. Except at this point, the tumour was sharply bounded from the brain tissue, which seemed to be pushed in front of it rather than invaded by it. Behind, the caudate nucleus was displaced about an inch backwards, but was not invaded by the growth.

Running sagittally forwards from the hinder end of the tumour was a yellow line indicating an almost complete antero-posterior fissure in the growth. On opening out the tumour at this point, it was obvious that this cleft represented the remains of the anterior cornu of the lateral ventricle which had been almost obliterated by the tumour which had been growing on both sides of it.

It therefore seemed clear that this tumour had its origin in the ependyma lining the anterior cornu of the ventricle; that it had grown forwards, pushing the white matter in front of itself and causing it to atrophy without infiltrating it appreciably; that it had similarly pushed backwards the caudate nucleus; that its further growth had obliterated the cavity of the ventricle in this region, and that at some later period it had burst into the lateral ventricle near the left foramen of Monroe.

No other large growth was found in the brain, but in view of the peculiar arrangement of the tumour mass around the spinal cord the ventricles of the brain were further explored. It was then found that several gelatinous masses were present, growing from the membranous lining of the ventricles of the central nervous system. The largest of these was found in the dependent part of the posterior cornu of the left lateral ventricle; it was a sessile and rather villous growth, which projected about 5 mm. into the ventricle. The choroid plexus in the same region contained a mass of growth about 6 mm. in diameter, but was not adherent to the other growth.

Scattered throughout the third ventricle, the aqueduct of Sylvius and the fourth ventricle were smaller masses, often—at the base of the fourth ventricle—forming a soft continuous layer of growth; the pineal body and the velum interpositum were much thickened by tumour mass.

On more careful examination of the thickened membranes, it was seen that the pia mater was more extensively infiltrated than the arachnoid. Although the latter was in many places adherent to the pia mater, in others it was only normally attached and preserved its naturally delicate structure. In other regions, as in the upper dorsal region of the cord (see fig. 1), the arachnoid was involved and adherent to the pia mater posteriorly, but was free on the anterior surface.

Microscopic Examination.

The growth in the frontal lobe is richly cellular, with a slight basis of neuroglia. The cells are spindle shaped with large oval nuclei and a comparatively small amount of protoplasm; they are arranged for the most part in whorls, which occasionally have a blood-vessel for their centre. Scattered irregularly through the tumour are found in considerable numbers large deeply-staining multinuclear cells.

The growth in the choroid plexus of the lateral ventricle showed a basis of fibrous tissue arranged more or less regularly like the stroma of a scirrhous cancer, each space containin groups

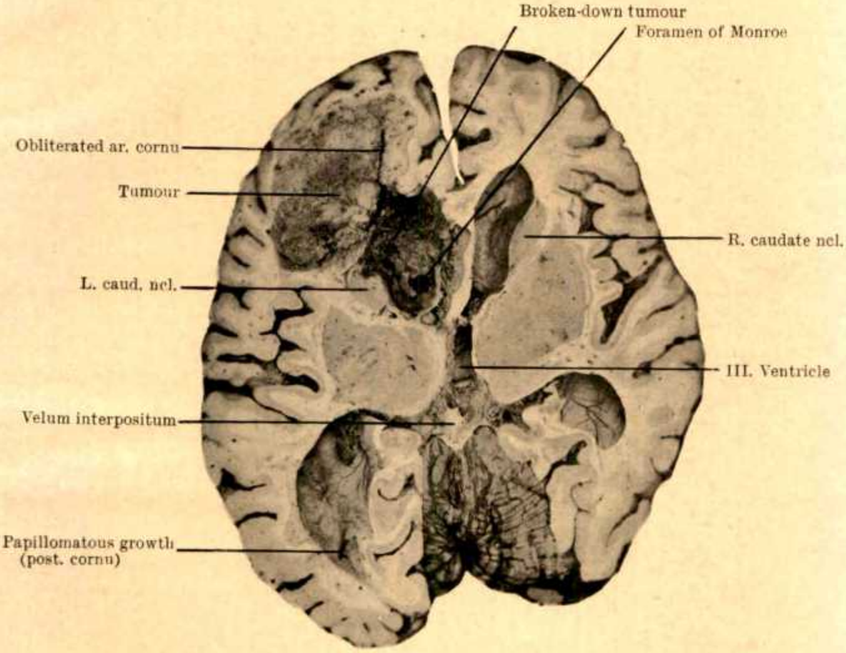


FIG. 2.

Horizontal section of brain of Case 1, showing the frontal tumour and point of rupture into foramen of Monroe.

of cells with comparatively large oval nuclei; the growth here is typical of alveolar sarcoma.

Around the cord the growth presents similar characteristics to those described above; the amount of fibrous tissue varies considerably in different parts, some being richly cellular and resembling the growth in the brain, whilst others are similar to that in the choroid plexus. The growth throughout is highly vascular. The emerging roots, particularly the posterior, are considerably invaded by the growth, which in many places has split up the rootlets into their constituent bundles, and has occasionally even separated the individual fibres from one another. Very few fibres seem to be destroyed altogether.

At the edge of the cord the growth is attached at short intervals by pial in growths; none of these seem to carry into the cord any of the special cells of the tumour. At the emergence of the rootlets, neuroglia fibres pass directly into the tumour, but are almost immediately lost in the fibrous stroma of the latter.

In the Gasserian ganglion, the growth is of the same character as in the choroid plexus. There is a large amount of fibrous tissue present, and the tumour cells are invading the ganglion along its interstitial tissue in such a way that there is a minimum of destruction with a maximum of infiltration. The posterior root ganglia show the same changes.

From several regions of the cord, sections were stained by the Nissl method, but no marked changes in the nerve cells were found. A few of the anterior horn cells showed early chromatolysis, and in some of them there is marked eccentricity of the nucleus, but none show more severe degeneration.

Stained by the Marchi method, many of the posterior roots showed considerable degeneration proximal to the root ganglia; the rather diffuse staining suggested that the degeneration was not all of the same age, but that some had been present for a considerable time.

The posterior columns showed marked ascending degeneration of a diffuse character, evidently derived from partial degeneration of most of the entering posterior roots. At the periphery of the cord, where the encircling tumour was attached, a few scattered degenerated fibres were seen; they did not appear to belong to any particular tract.

Stained by the Weigert-Pal method, there was well-marked diffuse degeneration of the columns of Goll and Burdach. All the posterior roots passing through the tumour seem to be partially degenerated, though not all to the same extent. The anterior roots are less affected.

Case 2.—H. H., aged 50, was admitted to the Queen's Hospital on February 1st, 1905, with pain in the abdomen. On admission, he was a well-developed man, and the symptoms he exhibited suggested early carcinoma of the stomach. He complained of no headache, but vomited occasionally, and there was no hydrochloric acid in the gastric juice after a test breakfast. An indefinite resistance in the epigastrium and an enlarged gland in the left supra-clavicular region were the only definite physical signs found. During the next few weeks he became curious in manner with hesitating speech and more persistent vomiting, although this was never severe. He died on February 26, 1905.

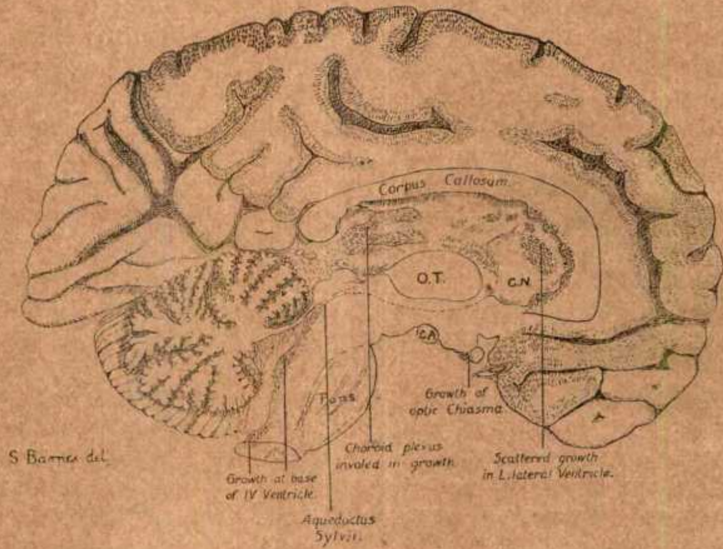

Corpus Callosum
O.T.
C.N.
C.A.
Pons
Growth of optic Chiasma
S Barnes del.
Choroid plexus involved in growth
Scattered growth in L. lateral Ventricle.
Growth at base of IV Ventricle
Aqueductus Sylvii.

FIG. 5.

Case 2.—Sagittal section of brain, a little to the left of the middle line. Masses of growth are seen on the walls of the lateral and fourth ventricles, and the optic tract has become invaded by extension through the floor of the third ventricle. The growth is shaded with small circles.

C.N. cauda nucleus; O.T. optic thalamus; C.A. corp. albic.

At the autopsy, held the following day, there was found a large mass of retro-peritoneal growth which was adherent to but which had not perforated the duodenum. The iliac, aortic, posterior mediastinal and supra-clavicular lymphatic glands were all enormously enlarged, and on microscopic section gave the appearance of alveolar sarcoma or endothelioma.

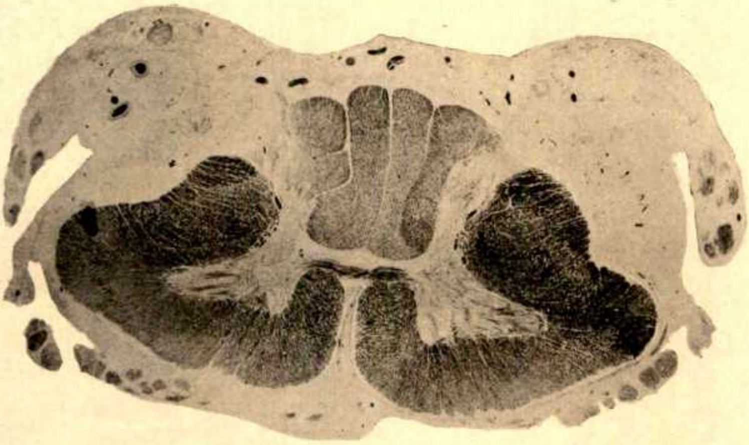


FIG. 3.

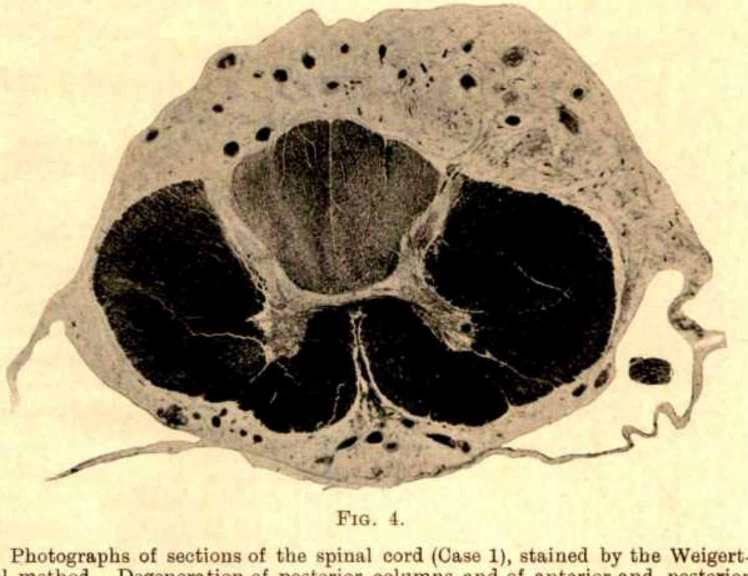


FIG. 4.

Photographs of sections of the spinal cord (Case 1), stained by the Weigert-Pal method. Degeneration of posterior columns and of anterior and posterior roots as they pass through the surrounding tumour. Arachnoid only partially involved.

The brain was then examined. On the right side the temporo-sphenoidal lobe contained a large diffusely infiltrating growth which invaded the tentorium cerebelli, and extended deeply so as to appear on the inner surface of the lateral ventricle near the foramen of Monroe. The surface of the growth facing the ventricle was extremely soft and ragged. On further inspection, it was found that the velum interpositum was also invaded by growth (by continuity), and that many discrete superficial masses of a similar nature were scattered over the surfaces of the lateral ventricles of both sides; the drawing (fig. 5) shows the inner aspect of the left lateral ventricle (sagittal section of the brain, just to the left of the middle line). The base of the aqueduct of Sylvius was free from growth, but the bases of the third and fourth ventricles contained several discrete masses tending in places to become confluent. At the base of the third ventricle in the middle line the lamina cinerea was infiltrated in its whole depth, and the growth had here invaded by extension the adjacent optic chiasma. Otherwise there was no sign of any involvement at the base of the brain.

The tentorial surface of the cerebellum on the right side immediately opposite the tumour in the temporo-sphenoidal lobe was covered with a thick milky adherent pia-arachnoid, which on section showed many cells indistinguishable from those of the growth, and which were not merely inflammatory exudation.

The spinal cord showed no abnormality, its membranes being normal throughout. Most of the posterior root ganglia were removed at the same time; all those of the cauda equina, and some of those of the dorsal region were found to be greatly enlarged—to about twice their normal size. The other ganglia of the cervical and dorsal regions were apparently normal. On macroscopic section of the enlarged ganglia the ganglion proper was seen to be intact, but the tissues around the point where the posterior root enters the ganglion were much matted and thickened.

Sections taken through the large growth in the brain showed masses of oat-shaped cells with very little stroma—a very simple type of growth, but evidently of great malignancy. The other masses on the lining of the ventricular wall showed a similar structure.

Several sections were taken through the spinal cord at various levels, but at no point could any definite meningeal growth be made out, the cord appearing when stained by the Van Gieson method to be normal.

Sections were then taken through the enlarged posterior root ganglia of the cauda equina. All of those examined showed well-marked aggregations of cells identical in form and size with those constituting the growth in the brain. These cells were arranged irregularly in groups, occupying the open lymphatic spaces immediately around the ganglia and the roots of the nerves, particularly the posterior, as they neared the ganglion from the cord. There was no sign that any of the nerve-fibres or cells were destroyed in any of the specimens examined. It was clear that there was an early infiltration of the root-ganglia by tumour cells, and there could be little doubt that their focus of origin had been the mass of growth in the cerebrum from which the metastases had been wafted by means of the cerebrospinal fluid.

Case 3 (Coupland and Pasteur).[1]—The patient was a single woman, 22 years of age. Her only previous illness had been rheumatic fever.

Her fatal illness began with bilious headaches and morning vomiting; pain in the shoulders and arms, and in the occipital region of the neck. Some delirium and motor weakness were soon added to her other symptoms, and it was then found that well-marked optic neuritis was present, and that both external recti were paralysed; the knee-jerks were lost. There were no convulsions.

Post mortem.—There was an excess of cerebrospinal fluid. The medulla and pons were invested with a jelly-like growth in which both sixth nerves were imbedded. The theca was adherent to the cord at one or two points. The cord was ensheathed in its whole length by a semi-transparent soft growth, which was most abundant on the posterior and lateral aspects. Many nodules were present on the intraspinal nerve-roots. Microscopically, the tumour was a small round-celled sarcoma.

Scattered growths were also found on the arachnoid at the base of the brain.

Case 4 (Coupland and Pasteur).[1]—After a fall down stairs, a girl, 4½ years old, became irritable, developed a squint, lost her power of walking, had pain in the head, and later optic neuritis. There was no anæsthesia or local paralysis. Difficulty in raising the eyelids, weakness of the trunk muscles and dilatation of the frontal veins followed. Before death, both arms were totally paralysed, but the feet never completely lost their power. There were no convulsions.

Post mortem.—There was increased intracranial pressure and excess of fluid in the ventricles. Beneath the arachnoid covering the inferior surface of the cerebellum was a layer of greenish material which penetrated into the nervous tissue for about half an inch. The middle lobe of the cerebellum was almost entirely replaced by soft vascular growth. Few scattered islets of tumour formed thin plaques at the base of the brain in the pia mater. Over the whole cord the pia was thickened posteriorly, the maximum thickness being about 6 millimetres. In places there was growth on the lateral and posterior aspects of the cord.

Microscopically, the growth was a soft sarcoma.

Case 5 (Ollivier)[2].—A boy, 11 years of age, had complained of attacks of headache accompanied by retraction of the head and recurring every six months. There was vomiting, pain in all four extremities, but consciousness was not disturbed, nor was there any anæsthesia or paralysis. Nausea, constipation, convulsions, and irregular breathing were also present.

Post mortem, a large growth was found in the cerebellum, whilst the whole spinal cord was enveloped, particularly on its posterior aspect, by a tumour-like mass of the consistency of brain-matter, which appeared to blend laterally with the arachnoid.

Case 6 (Schultz)[3].—The patient was a girl of 16, with a strong neuropathic history. Her chief symptoms were pain in the back, tingling in the hands, and gradually increasing weakness of the arms; later, tingling and weakness came on in the legs also. Headache then came on, but there is no note as to the optic discs. There was rigidity of the spine, and before death the right knee-jerk was lost, whilst the left was weak.

Post mortem, the cranial contents and their membranes were healthy. The sub-arachnoid space from the cervical region to the cauda equina was occupied by a mass of new growth covered by normal arachnoid. The new growth was nodulated and contained hæmorrhages; it ensheathed the cord (excepting in part of the dorsal region), but was more abundant on the posterior aspect than on the anterior.

Microscopically, the growth was an alveolar sarcoma, probably originating in the pia mater.

Case 7. (Hughes Bennett).[1]—The patient was a man, 48 years of age, whose illness began with shooting pains in the legs. The legs then became ataxic and weak, and soon similar defects were noticed in the arms. Both knee-jerks were lost; sensation in the feet was retarded and dulled, but elsewhere was good. There were no ocular signs. Death from uncontrollable vomiting.

Post mortem, there was some flattening of the convolutions. The choroid plexuses of the lateral ventricles were œdematous, each lateral ventricle containing 4 cc. of clear yellow fluid. "In the substance of each frontal lobe, involving the anterior cornua of the lateral ventricle, was a circumscribed patch of softening, on the left side about the size of a hen's egg, and on the right side somewhat smaller. These extended inwards to the third ventricle and longitudinal fissure, downwards to the base of the brain and upwards to within one inch of the cortical substance." Microscopically, this area gave the appearances of cerebral softening.

The spinal dura was free, but the pia was thickened especially on its posterior aspect, most markedly below and gradually diminishing from below upwards. Posteriorly, the pia was studded with numerous tumours, more numerous below where they became confluent. The growths were attached to, and surrounded the posterior nerve roots. One sarcomatous mass was present in the floor of the fourth ventricle, and others were found on the under surface of the cerebellum.

Microscopically, the growth was a round-celled sarcoma.

Case 8. (Heubner).[5]—The patient was a girl with a good previous history. A short time after a fall on the arm, she began to experience cramps in the upper extremities, stiffness in the neck, headache, vomiting, disturbance of consciousness, and rapidly progressive loss of eyesight; hydrocephalus came on and steadily increased; there was pain in the upper arms, and flaccid palsy of the lower extremities with loss of knee-jerks at a late stage.

At the autopsy, there was a marked hydrocepalus. The membranes of the brain and spinal cord were greatly thickened, and six separate tumour masses, afterwards found to be gliomata, were present in the dorsal region of the cord. Secondary degeneration in the posterior columns was present.

The author concludes that the gliomata of the cord developed first, and that there was a secondary meningitis spreading up to the brain with a resulting hydrocephalus. He admits the difficulty of supposing a primary cord lesion, with symptoms which at the onset were purely cerebral.

Case 9. (Pels Leusden).[6]—In December, 1893, a man, aged 22 years, began to suffer from "rheumatic" pains in the back and legs; these gradually increased, and then the left leg became stiff and weak. Paralysis of the right leg followed, and then he began to complain of headache, intercostal neuralgia, and failing vision. By May, 1895, he had also difficulty in micturition and defæcation, and on admission to hospital he was very cachetic. There was now found to be complete paralysis and anæsthesia of both legs; a slight convergent strabismus was present. In the right eye, there were opaque nerve fibres at the edge of the optic disc, and it was impossible to be certain whether optic neuritis was present, but it was undoubtedly present in the left eye.

Post mortem, a tumour was found infiltrating the pia-arachnoid at the base of the brain and around the spinal cord, occupying practically the same positions as that figured in Case 1. Microscopically, the growth was a glia-sarcoma.

Case 10. (Fischer)[7].—An eight-year old girl began to have a girdle pain in the abdomen, and in the small of the back, and weakness of the legs. When examined in June, 1899, there was complete flaccid paraplegia, paralysis of the bladder, and complete loss of all forms of sensation in the legs and in the trunk below the fifth dorsal root on the right, and the seventh on the left. There was no pain on percussion over the vertebræ.

In November there was commencing weakness of the upper extremities, and these were completely paralysed by February, 1900. The right pupil was now larger than the left, and there was congestion of both optic discs with tortuosity of the veins. Death occurred in March, 1900.

At the autopsy, the fifth to the tenth vertebræ were attached to the spinal cord which was greatly thickened. In the lumbar region a growth had made its way forwards through the bodies of the vertebræ to form a lump in the abdominal cavity the size of a hen's egg. The whole of this region of the cord was replaced by growth. The medulla, the base of the fourth ventricle, and the corpora quadrigemina were all infiltrated with growth. In the base of the third ventricle between the optic tracts was a translucent greyish neoplasm, whilst a second similar one was present in the left hemisphere, involving the head of the caudate nucleus. The growth was a glia-sarcoma, and the author considered that the growth in the cord was the primary one.

Case 11. (Pfeiffer)[8].—The patient was a 64 year old woman, with no inherited tendencies. For six months had suffered from cough with muco-purulent expectoration, wasting, no hæmoptysis, no night-sweats. At the same time, pains were present in the outer side of the right thigh spreading down to the foot.

On first admission (March, 1889), pulmonary tuberculosis was found at both apices, and there was present sciatica on the right side. Otherwise normal.

On second admission, three months later, besides the pulmonary condition there was present right-sided optic atrophy, and the right knee-jerk was absent; soon afterwards the left knee-jerk was also lost. A clinical diagnosis of pulmonary tuberculosis and incipient tabes was made.

Post mortem, there was found in the nervous system a "tuberkel im linken Corpus caudatum." There was great thickening of the arachnoid, of the extra-medullary nerve-roots of the spinal cord, and infiltration of the sheaths of the optic nerves, especially of the right, with epithelial cells. There was hyaline degeneration of many of the blood-vessels, and, by the Weigert method, degeneration in the posterior columns of the cord. There were also irregular deposition in and around the medulla. There is no report of the microscopic characters of the tumour in the caudate nucleus, nor was the cerebrum examined. In discussing the case, the author admits the strong resemblance of the epithelial formation to new growth, but on account of its widespread situation, concludes that it is a congenital anomaly. He remarks that the cylindrical epithelium reminds one forcibly of that of the central canal, and thus supports the idea of a congenital abnormality.

In many of the cases here recorded it will be noticed that a tumour mass was present which more or less ensheathed the whole spinal cord, whilst in some of the cases the meninges at the base of the brain were also infiltrated with sarcoma. In recording their work, most of the authors have attempted to fix upon some point as the origin of the growth. Coupland and Pasteur, in their summary conclude that the seat of origin in all the cases they considered (viz., Cases 3, 4, 5, 6 and 7) was spinal, except in the case reported by Ollivier (No. 5), which they consider to have been primarily cerebellar. They assume that the growth spread by continuity from the spinal pia both upwards and downwards, thus eventually involving the pia at the base of the brain late in the disease. Heubner (Case 8) also insists upon the primary spinal origin of the tumours in his case, although all the clinical symptoms seemed to show that the disease in the brain arose first. Fischer (Case 9) also considered that in his case the disease was primarily spinal, and that the cerebral membranes were secondarily infected, but he does not give any adequate explanation of how this occurred. It seems to me that it is far more likely that in most, if not in all these cases, the primary growth was situated high up in the nervous system. In my cases it is quite clear that the primary growth was in the frontal lobe of the brain, and that a rupture of the tumour had caused infective particles to become disseminated throughout the central nervous system by means of the cerebrospinal fluid. In this way it is easy to trace the path of the infective particles flowing with the stream instead of against it, as would necessarily be the case if the growth originated in the spinal pia. If we assume that the cerebrospinal fluid secreted by the choroid plexuses of the lateral ventricle became charged with tumour particles, each capable of continued growth in a suitable nidus, it is then easy to understand how the whole track of the fluid down the ventricular system of the brain and in the subarachnoid spaces of the brain and cord would become the seat of a mass of secondary sarcoma. On this assumption, the solid particles would be filtered off from the fluid as it left the cerebrospinal canal, particularly along the course of the posterior roots near the ganglia, whilst other particles would be left more or less regularly in the lining of the ventricles of the brain and in the subarachnoid space around the cord. In each region the subsequent development of the growth would give rise to the appearances which have been described.

A strong argument in favour of the intracranial origin of these masses of growth is furnished by the analogous condition of tubercular meningitis. In a case of tubercular "tumour" which recently came under observation, in which death occurred from tubercular meningitis, it was noted that the involvement of the membranes at the base of the brain and of the pia mater of the cord corresponded exactly to that of the growth in Case 1. Here the primary focus was a "tumour" (tubercular), about the size of a hazel-nut, on the exposed under surface of the cerebellum, and, though it had not ruptured, it was clearly the point of origin of the meningitis. There could in this case be no question of spread by continuity from below: the infection was clearly one which spread by the cerebrospinal fluid.

It may be argued that in the first case which I record (Case 1) the rupture of the tumour may have given rise to the secondary growths, but that without rupture no such dissemination would have occurred. Again I fall back on an analogy. In those cases of disseminated carcinoma of the peritoneal cavity, where the secondary growths are small and scattered (not continuous) over the parietal and, to a less extent, over the visceral peritoneum, there can be no doubt that the original focus has infected the peritoneal fluid, and that the dissemination has occurred by the streaming of these particles of malignant growth towards the stomata, where they are arrested and form new growths. And yet in these cases it is the exception rather than the rule to find a carcinomatous gland, or other tumour, which has actually ruptured. It may be that the rupture has been a small one, and that it has healed before the death of the patient.

The main argument against this mode of origin and distribution has been that a large growth has not always been found in the brain. Such a growth was present in my first case in such a marked degree that the most sceptical could hardly doubt that it was primary. In Case 4 there is a suggestion that the primary growth was in the vermis of the cerebellum roofing in the fourth ventricle; in Case 5, again, the primary growth was in the cerebellum; in Case 7, which differs from the others in that the growths did not become universally distributed over the spinal cord, but in no other essential particular, it seems to me more than likely that the softening in the frontal lobe was in reality a softened primary growth, and that this softening was the direct cause of the dissemination. In Case 8 I find it difficult to believe that we are dealing with a true meningitis spreading up to the brain as a result of the tumour.

The difficulty of distinguishing chronic inflammatory tissue from sarcoma is such that the microscope alone is not always sufficient, and whatever the histological appearance one would be inclined to think that the "meningitis" in this case was in reality a diffuse sarcomatous infiltration of the membranes.

In most of the recorded cases, the growth around the cord, though often coarsely uneven in thickness, does not suggest that at one point it has been present for a long time before growth occurred elsewhere; with one exception (Case 10) the spinal cord is not destroyed at any point, nor is there any great single mass of growth in the spinal region.

It, therefore, seems far more rational to look further afield for the point of origin of the tumour, and to see if the spinal mass is not all of the same age, and secondary to some other growth.

All of the cases are of a sarcomatous nature, but they vary considerably in malignancy. It is one of the most remarkable features that despite their almost complete involvement of the spinal cord, they rarely invade its substance, and then only to a very slight extent. The sarcoma seems to have a special affinity for the pia mater, growing easily amongst its tissue spaces, but not easily bursting through the membrane to invade the cord. In one of the cases, this invasion had occurred in several segments of the cord, and Fisher, therefore, concluded (Case 10) that this was the point of primary growth; that this growth was one of unusual malignancy is perfectly clear, but I am much inclined to doubt whether the growth was primary in the membranes of the spinal cord. It is difficult on that assumption to see how a growth could have occurred, as is described, in the head of the caudate nucleus, and how the medulla and mid-brain could be involved in growth on their ventricular surfaces. It could not be by dissemination through the cerebrospinal fluid, for the flow is in the downward direction, and such a spread would have to be upwards; so that the only way remaining would be by continuity—and the continuity is obviously frequently broken.

One objection may be raised to the suggestion that these growths have an intracranial origin, viz., the absence of intracranial symptoms until late in the disease. It must be remembered, however, that the primary growth usually seems to originate in the ependyma lining of the ventricles, the third and lateral particularly, and that if, for instance, the caudate nucleus were the point of involvement, we should expect no symptoms until the growth had attained a considerable size, and had begun to give rise to the general signs of intracranial tumour. But in such a position, with the cerebrospinal fluid washing directly over the growth, it is highly probable that dissemination would occur before the primary growth had given rise to any symptom of its existence, and it might easily happen that the first signs should be shown by the secondary growths.

The relative non-malignancy of these growths is also shown by the way in which the intraspinal nerve-roots are affected; as a rule, there is much infiltration of the various bundles of fibres, and even separation of individual fibres, without marked destruction of the latter. It becomes an interesting speculation as to whether there do not exist types which are still malignant in the sense that they give rise to metastases, but which are less rapid in their growth and exhibit more innocent histological characters. It is possible that some of the cases of central intracranial and intra-vertebral neuro-fibromatosis are of this nature. The situation of the nodules of growth in these cases is similar—chiefly on the nerve-roots (especially of the cauda-equina), and less frequently on the pia of the spinal cord. It is true that we usually denominate them as "innocent" growths, and yet, single growths arising in the posterior root sheaths are known to recur locally if on removal a fragment of the root-sheath is left behind. The danger of recurrence under a malignant form after operative interference has so often been remarked upon that one cannot help thinking that the original growth has at least potential malignant characters, and that it may happen that in the course of its growth it may at times disseminate particles which can grow in a suitable habitat. For such growths, it seems clear that there is a special predilection for the sheaths of nerves, and that even were the infective particles to be wafted to the lungs or liver, they might be unable to grow there on account of unsuitable surrounding conditions. It is thus possible that certain of the cases of central neuro-fibromatosis may be regarded as being the result of dissemination of secondary infective particles from a single neoplasm higher up in the central nervous system, rather than as the expression of a series of congenital malformations.

Another point of interest in these cases has been the diffusion of the growths in the ventricles of the brain. In Cases 1, 2, 10 and 11 there were present discrete masses of growth in the lining of the third, lateral and fourth ventricles in none of the cases was the growth very obvious, and it is possible that similar growths have been present, but have not been recognised in some of the other cases. The occurrence of such growths in Cases 1, 10 and 11, in association with involvement of the arachnoid and pia around the spinal cord can hardly be regarded as a mere coincidence, and it seems only rational to suppose that both have occurred as metastases from some primary growth. Thus, in Case 1 the tumour in the frontal region could give rise to particles which might infect the ventricles, whilst other particles might easily pass with the cerebrospinal fluid through the foramina in the roof of the fourth ventricle, and so reach the cisterna magna, the subarachnoid spaces of the spinal cord, and, finally, the posterior root ganglia, where the last of such infective particles would be filtered off. In Case 10 it is easy to understand the course of the disease if we assume that the growth at the base of the third ventricle, or that in the caudate nucleus, was the primary one, and that one of the early metastatic growths grew sufficiently rapidly to give rise to a large tumour at the time of death; whilst a growth in the caudate nucleus in Case 11 would adequately explain the method of origin of the various growths found at the autopsy.

From a clinical point of view, the symptoms described have naturally been very varied, partly on account of the disseminated character of the growth, and partly because in some cases there has been an obvious growth, or in some cases several growths, within the brain. In any case there is almost constantly present in the later stages some hydrocephalus, clearly secondary to the growths. This may be caused either by blocking of the outlet from the skull of the cerebrospinal fluid or by blocking of a large number of the possible outlets from the spinal theca. It will therefore be seen that the headache, vomiting, and optic neuritis, if they occur at all, may either arise at an early stage before the symptoms due to large local growths have shown themselves, or may only be present as the terminal phase of the condition

Considerable discussion was devoted by Coupland and

Pasteur to the question as to whether convulsions should be expected, and they thought that the absence was one of the most characteristic phenomena of the condition. But, from a consideration of the other cases, it is clear that they do occur, although not in the early stages. This is probably due to the fact that the cerebral cortex is not as a rule directly involved, the growth tending to affect the ventricular lining and the pia mater rather than the cerebral substance. The convulsions which do occur are probably in most cases due to the rise in intracranial pressure which comes on late in the disease, although in Case 1 they may have originated in the diseased frontal cortex.

It will be noted that peripheral pain along the area of supply of the involved nerve roots was present in most of the cases, but rarely reached a severe grade such as is usually seen in single root tumours. This may be due to the fact that the nerve roots are not severely compressed by the rather soft encircling growth as they are by the less malignant and often fibrous single tumours. It is, however, more likely that the youth of the patients is a better explanation, for the only middle-aged patients in the group suffered so severely from shooting pains that they presented the clinical picture of tabes dorsalis.

In general it remains impossible to give any accurate diagnostic signs of the condition; but it seems clear that if a patient who is known to present the signs of unlocalisable cerebral tumour develops well-marked pains in the limbs and trunk, with early loss of deep reflexes and rapid wasting, the presence of disseminated growth in the spinal pia and posterior root ganglia should be suspected. In such cases it would be well to examine also the ventricular lining in case the less obvious gelatinous growths which occur in this situation may be overlooked.

CONCLUSIONS.

(1) If a growth originates in the caudate nucleus or in any other part of the brain and eventually finds its way to the lining of the ventricular cavities, it may "infect" the cerebrospinal fluid. Metastatic growths may then occur in the

following situations: (1) In the lining walls of the lateral, third or fourth ventricles; (2) In the meninges (subarachnoid) at the base of the brain; (3) In the pia mater and arachnoid around the spinal cord; and (4) in the posterior root ganglia, particularly of the cauda equina.

(2) In all probability diffuse sarcomatous infiltration of the spinal pia-arachnoid, which is occasionally found post-mortem, is in all cases the expression of a sarcomatous infection of the cerebrospinal fluid as a result of some primary growth which lies exposed to the stream of the cerebrospinal fluid high up in the nervous system.

(3) The regions occupied by such secondary growths are exactly the same as those inflamed by tubercular meningitis after the rupture of a tubercular "tumour" of the brain.

Bibliography.

(1) Coupland and Pasteur. *Trans. of Path. Soc.*, xxxviii., p. 26.
(2) Ollivier. "Traité des Maladies de la Moelle Epiniere" (1837, T. 2, p. 490).
(3) Schultz. *Arch. f. Psych.*, 1885, p. 542.
(4) Hughes Bennett. *Clin. Soc. Trans.*, vol. xviii., p. 168.
(5) Heubner. "Ueber ein Fall von Multiplen Rückenmarksgliomen, Meningitis und Hydrocephalus." *Verhand. der Gesell. für Kinderheilkunde zu Aachen*, 1900.
(6) Pels Leusden. *Ziegler's Beiträge*, Jahrg. xxiii., S. 96.
(7) Fischer. "Ueber ein selten mächtig entwickeltes Glia-sarcomatodes des Rückenmarks." *Zeitschrift f. Heilkunde*, xxii.
(8) Pfeiffer. "Ueber eigenartige Veränderungen in der Arachnoides, der extramedullären Rückenmarkswurzeln und der beiden Nervi optici." *Deutsche Zeit. für Nervenheilkunde*, 1894, B. 5, p. 45.

SOME HITHERTO UNDESCRIBED SYMPTOMS IN ANGINA PECTORIS.

BY G. A. GIBSON, M.D., D.SC.EDIN., LL.D.ST.AND., F.R.C.P.EDIN.

A Clinical Lecture delivered in the Royal Infirmary.

ALTHOUGH angina pectoris was the subject of one of my clinical lectures last term, the subject is so important as to merit further study. We have, moreover, at present in our wards the most interesting example of the affection which it has ever been my good fortune to observe. The complex of symptoms presented by this patient is so extraordinary that it seems my duty to lay the facts before you in full detail. They will furnish an occasion for amplifying the remarks previously made on the modern conception of the condition.

A labouring man, aged 45, born in Leitrim, but resident for some years in Edinburgh, was admitted to my ward on account of pain in the chest and left arm. His father lived to the age of 85, and was said to be subject to rheumatic pains. His mother died, when 33 years old, of puerperal causes. The patient says he was never addicted to alcoholic excess, seeing that for him it was too expensive a luxury. He has been in the habit of smoking about two ounces of strong tobacco every week. His home conditions are healthy and comfortable, but his work is rather severe. When 21 years old he suffered from some fever, as to the nature of which he is ignorant. In more recent years he had an attack of pneumonia. The commencement of the present illness appears to have been about six weeks ago, and it seems to have begun with breathlessness on exertion; the actual onset of the pain in the chest and arm was five weeks before admission. Its first appearance was when he was in a train on the way to his work. He was able to perform his duties for three days, but since then has been unfit for any exertion. The pain has persisted continuously ever since its first onset, but two weeks after it began it underwent a change. At first it was confined to the region of the left scapula, but it afterwards began to shoot down the left arm, and at the same time it showed paroxysmal exacerbations.

The present condition of the patient is as follows:—He is a strongly-built man, 5 ft. 7 in. in height, weighing 10 st. 1 lb., with a healthy, florid complexion.

The circulatory system gives evidence of considerable alteration. The patient, as a rule, has a high complexion, except during a paroxysm of pain, when he becomes paler. There is some dilatation of the venules on the cheeks and nose, and the temporal arteries stand out in a sinuous fashion, although there is no excessive pulsation in them or in the carotids. The superficial arteries are thick and tortuous. The arterial pressure is high at all times; measured by Hill and Barnard's sphygmometer it varies between 160 and 170 mm. Hg. During the paroxysms of pain the pressure rises considerably, but for obvious reasons it is then difficult to obtain an exact measurement. The pulsation is perfectly regular and not unduly frequent; each individual pulsation

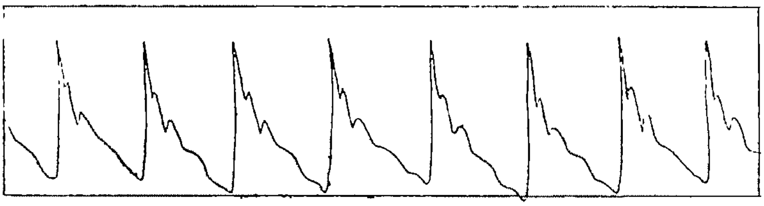


FIG. 1.
Usual pulse. Pressure 4 oz.

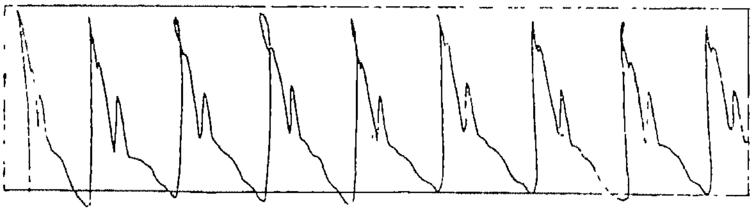


FIG. 2.
Usual pulse. Pressure 5 oz.

is of large amplitude and is well sustained. Tracings of the ordinary pulse are given in figs. 1 and 2.

The Junior House Physician was in the act of taking some sphygmographic tracings when one of the paroxysms of pain set in, and he found that with the increase of pressure there was a characteristic reduction in the size of the pulse, with some acceleration, as shown in fig. 3, while, after the use of nitrite of amyl and the relief of pain, the pulse became large, bounding, and still more frequent, as in fig. 4. The case, in respect of the pulse, therefore, conforms to the type of angina with spasm of the arterioles.

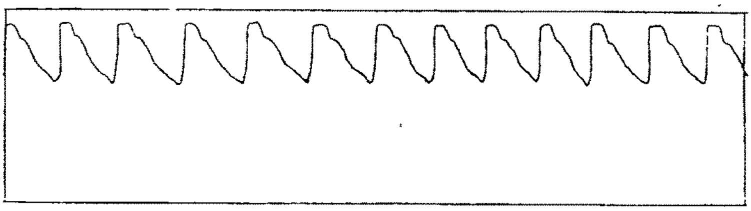


FIG. 3.
Pulse during paroxysm of pain. Pressure 3 oz.

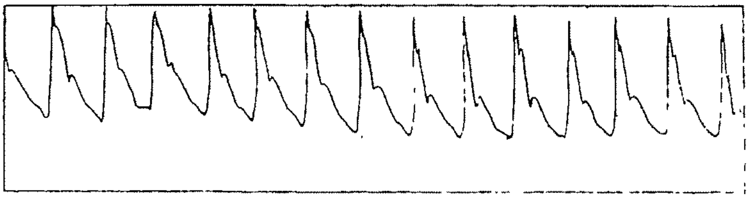


FIG. 4.
Pulse after nitrite of amyl. Pressure 3 oz.

On inspection, the apex beat is seen in the fifth intercostal space, about the mammillary line. There is no other impulse visible in any part of the præcordia or epigastrium. On palpation, the apex beat is found to be situated 4½ in. from mid sternum. The impulse is forcible and without any thrill. The superior border of the heart on percussion is found at the upper edge of the third left costal cartilage.

The left border is 4¾ in. and the right is 2 in. from the middle line. The heart sounds are perfectly clear, but the aortic second sound is much accentuated.

The most careful investigation of the chest fails to give any evidence of other disease, and the X-rays, employed both by screen and by skiagraphy, show a perfectly normal state of matters at the base of the heart.

The patient complains of an extensive and constant pain, most severe, as a rule, over the back, where it extends down as far as the ninth intercostal space. It reaches up to the back of the neck, and passes round its side so as to reach the left clavicle, which it crosses, and extends down the middle line to the eighth intercostal space, from which point it becomes continuous with the pain at the back by passing round the inferior axillary region. From the shoulder it extends down the outer aspect of the arm and forearm so as to reach the thumb, index and middle fingers. The exact boundaries of the pain can be seen in the figs. 5 and 6, drawn from photographs taken after the areas had been marked on the skin. When the paroxysms of pain occur, the boundaries appear to the patient to be somewhat enlarged, and it

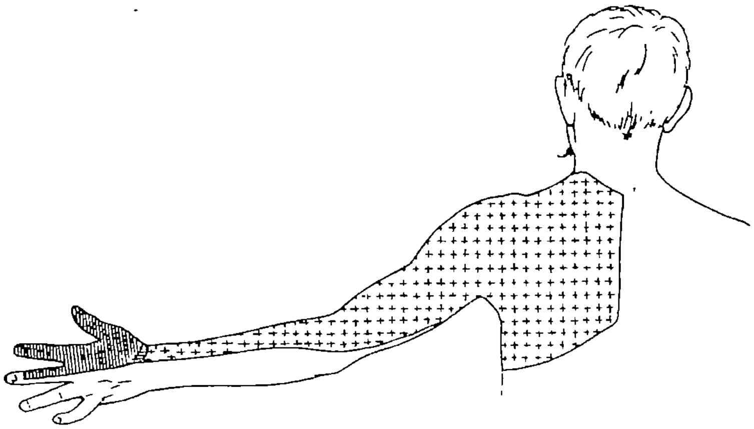


FIG. 5.

Distribution of sensory changes posteriorly. The crosses denote hyperæsthesia; the horizontal shading analgesia without anæsthesia; the vertical shading analgesia with anæsthesia.

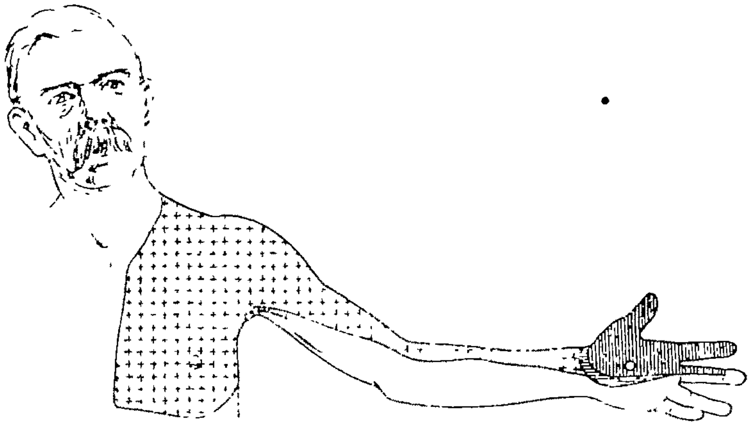


FIG. 6.
Distribution of sensory changes anteriorly. Explanation as in fig. 5.

shoots up the left side of the head so as to involve the cranial vault as far as the middle line, and includes most of the arm and forearm.

On testing the sensibility of the surface, a most interesting group of phenomena can be discovered. Over the whole

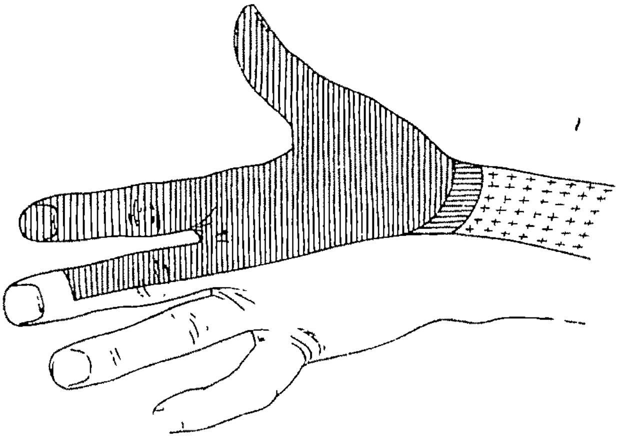


FIG. 7.
Sensory changes in back of hand. Explanation as on fig. 5.

Now, before leaving the afferent nervous system, it is necessary to point out two divergences from the usual state of matters exhibited by our patient. One of these may be due to an anatomical peculiarity. It is the line of division separating the insensitive from the sensitive part of the palm. Instead of being bounded by the middle of the ring finger, as is usual in front, the line of demarcation of ulnar and median nerves runs along the middle of the middle finger, unless we may assume that a portion of the median nerve has escaped. The other departure from what is customary is much more difficult to understand. As will be observed from the outlines painted on the patient, the inner or ulnar aspect of the arm, forearm, and hand, is not affected, *i.e.*, a considerable area belonging to the segmental distribution of the seventh and eighth cervical and first dorsal portions of the cord is intact, while the areas above and below are deeply involved. Now the segments unaffected in our case are those usually most deeply involved. In view of the cilio-spinal effects about to be described, this fact is difficult to understand, and it seems to me to indicate a capriciousness of impression on the part of the spinal segments.

The organic reflexes are normal. The cutaneous reflexes are all present and appear to be within usual limits. The supinator and triceps jerks are increased in the left arm as compared with the right, and the myotatic irritability is increased in the deltoid and the other muscles on the outer aspect of the left arm and forearm. There is great weakness of the left hand, especially in the thumb and first and second fingers, so that the patient's grasp is practically restricted to the ring and little fingers. With the dynamometer the grasp of the right hand is 135 and of the left 50. There is wasting and loss of tone in all the muscles of the shoulder-girdle, arm, forearm, and hand on the left side. The right arm is in circumference 12 in., the left 10½ in. The right forearm is 10½ in., and the left 10 in. On testing the electric reactions an increased excitability is found in the muscles which are affected. The exact state of matters will be easily understood from the following table, in which the measurements of faradism are expressed in the number of centimetres on the scale of the induction apparatus, while those of galvanism are given in milliampères:—

Faradism.

	R.	L.
Deltoid	2.7	2
Biceps	1·6	1·4
Supinator longus	1·9	1·2
Flexor sublimis	3·2	1·9

Galvanism.

	R.	L.
Deltoid	9 ccc. 10 acc.	6 ccc. 7 acc.
Biceps	5 ccc. 6 acc.	3 ccc. 4 acc.
Supinator longus	6 ccc. 7 acc.	5 ccc. 6 acc.
Flexor sublimis	11 ccc. 12 acc.	9 ccc 10 acc.

One other point deserves consideration. The left eye is more prominent than the right, and the left pupil is larger than its fellow. This condition has been present throughout his residence in the ward, but it is more marked at those times when the pain gives him serious trouble. There cannot be a doubt, therefore, that there is irritation of the sympathetic nerve on the left side, involving the cilio-spinal fibres. It is interesting to observe that these phenomena are not accompanied by any alteration in the functions of the skin. There is no redness or increase of perspiration, but, as already mentioned, there is some general pallor of the whole countenance during an attack.

The diagnosis in this interesting case is early arterial sclerosis, probably implicating the coronary arteries, and accompanied by some interstitial changes in the myocardium —the combination, in short, which is so commonly attended by angina pectoris. But, as already remarked, there is a vaso-motor element in the attacks; this, as is universally recognised, is a symptom frequently met with.

Numbness of the arm, the forearm, or the hand, has often been observed in cases of angina pectoris, but complete anæsthesia, such as is present in this case, must be rare indeed; in fact, no instance even distantly approaching that just described, has ever come under my notice. Neither in the beautiful researches of Mackenzie,[1] nor of Head,[2] is there any reference to such conditions. In a private communication, however, the former states that he seemed to obtain anæsthetic areas when he first investigated the sensibility in angina pectoris, but that in late years he had failed to find them and concluded he had made a mistake.

The wasting of the muscles has been described both by Eichhorst[3] and myself,[4] and there is no difficulty in explaining such trophic results from powerful afferent impulses. They are strictly analogous to the conditions which are found in arthritic muscular atrophy. This has long been considered as due entirely to reflex influence, which has been fully discussed by Paget[5] and by Vulpian,[6] and the theory is strongly supported by the fact that in experimental joint lesions wasting of the muscles is prevented by previous division of the posterior spinal roots, which has been described by Raymond.[7] But there are some distinct differences between the wasting in this case and in that of arthritic muscular atrophy. In this patient, just as occurs in arthritic atrophy, there is considerable increase in myotatic irritability. But no case of arthritic dystrophy of muscles has ever been under my notice in which there was an increase in the electric irritability of the atrophied muscles. It is possible, however, that what we are dealing with is an early condition, and that a reduction in electric irritability may be a later phase.

In addition to providing a number of hitherto undescribed symptoms for our study, the patient seems to me of the deepest interest as giving proof of a means of communication between the heart and the nerve centres which has hitherto received but scant attention.

---

[1] *Medical Chronicle*, 1892, p. 293; and *Lancet*, 1895, Vol. I., p. 16.

[2] BRAIN, 1893, vol. xvi., p. 1, and 1894, vol. xvii., p. 339.

[3] "Handbuch der Specieller Pathologie und Therapie," 1895, S. 231.

[4] "The Nervous Affections of the Heart," 1904, p. 18.

[5] The *Lancet*, 1873, Vol. II., p. 727.

[6] "Leçons sur l'Appareil Vaso-moteur," 1875, Tome 2, p. 327.

[7] *Revue de Médecine*, 1890, p. 374.

While the vagus centripetal mechanism has been long recognised, that of the sympathetic system has been a matter of great doubt. In my Morison lectures,[1] the course of these tracts was fully described and the conclusions then reached may be now briefly summarised. Certain channels, proved by undoubted clinical facts to supply the course of afferent impulses, have not yet received support from any experimental evidence. The afferent impulses passing upwards in connection with the inhibiting and dilating mechanism are carried by the vagus nerve. The afferent impulses belonging to the accelerating and constricting mechanism must travel by the inferior cardiac nerve to the inferior cervical ganglion and thence by the gray rami to the seventh and eighth segments of the cord. It is possible that they may also pass round the annulus of Vieussens to reach the upper thoracic segments. But of afferent nerves belonging to the heart and passing to the spinal cord, there is no experimental proof. If we may judge afferent nerves by those of the visceral system of the abdomen, they are always medullated up to the posterior ganglion, and many of these medullated fibres are of large size. Reasoning by analogy it might be expected that the afferent cardiac fibres must also be medullated, but Gaskell,[2] when working at the fibres of the dog's heart was much struck to find that all the nerves from the annulus of Vieussens to the heart were non-medullated—no medullated fibres were traced to the depressor or the vagus nerve. He therefore concluded that the anatomical evidence confirmed the physiological, and that both pressor and depressor fibres reached the central nervous system by the vagus nerve. By means of a private communication he has, however, expressed his opinion to me that the spinal heart nerves are only a special example of the system of vaso-constrictor nerves, and therefore, if the blood-vessels possess afferent nerves, the heart, in virtue of its connection with that system must possess such nerves. He believes that there is evidence of the possession of spinal afferent nerves by the blood-vessels, while there is no evidence to prove that the blood-vessels possess any number of medullated fibres. His view is accordingly that it is possible, indeed probable, that afferent nerves, at least from the coronary vessels, should accompany the accelerator and so account for the clinical facts.

---

[1] *Op. cit.*, p. 32.

[2] *Transactions of the International Medical Congress, London*, 1881, vol. i., p. 254; *Philosophical Transactions, London*, 1883, vol. clxxiii., p. 993; *Journal of Physiology*, 1884, vol. v., pp. 46 and 362; *Ibid.*, 1886, vol. vii., p. 1.

The real importance of this case, then, in my opinion, lies in the opportunity which it affords of giving a clear demonstration of certain facts regarding the afferent impulses from the heart; the symptoms exhibited by the patient allow the possibility of tracing out the course of these paths. In the diagram, fig. 9, the direction of these channels of communication is traced out. Passing up from the receptive end organ (*H*), by the cardiac nerves to the cervical ganglia (I *G*, II *G*, and III *G*), of the sympathetic, they pass in, as has been elsewhere shown by me,[1] by the gray rami communicantes (*PC*), to the posterior spinal roots, and thence run upwards in the ascending tracts of the spinal cord. In the cortex cerebri (*C*), the impulses which are produced give rise to sensation, but, as Head[2] puts it, "the sensory and localising power of the surface of the body is enormously in excess of that of the viscera, and thus by what might be called a psychical error of judgment, the diffusion area is accepted by consciousness and the pain is referred on to the surface of the body instead of on to the organ actually affected." The result is that the patient seems to feel uneasy or painful sensations over the præcordia, shoulder and upper extremity, as at *S*. That the cervical sympathetic is involved in this case cannot be denied on account of the ocular phenomena, and our patient may be taken as a proof of the contention that this system is the means of conducting afferent impulses from the heart. The figure (fig. 10) gives a diagrammatic representation of part of the cilio-spinal mechanism, showing the course of the spinal fibres

---

[1] *Op. cit.* p. 30.
[2] *Op. cit.*, vol. xvi., p. 126.

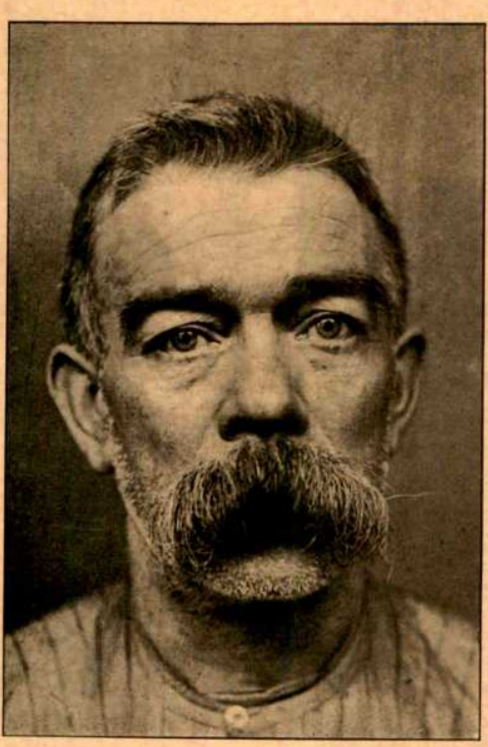


FIG. 9.

From a photograph of the patient, showing prominence of the left eye and dilatation of the left pupil.

from the pupil-dilating centre (*PD*) in the base of the brain down to the cilio-spinal centre (*CS*), and thence by the anterior spinal root and anterior or white ramus communicans (*AC*), to the sympathetic system, whence the fibres pass upwards to the radiating fibres of the iris (*DP*) to the muscle of Müller (*MM*), and to the non-striped

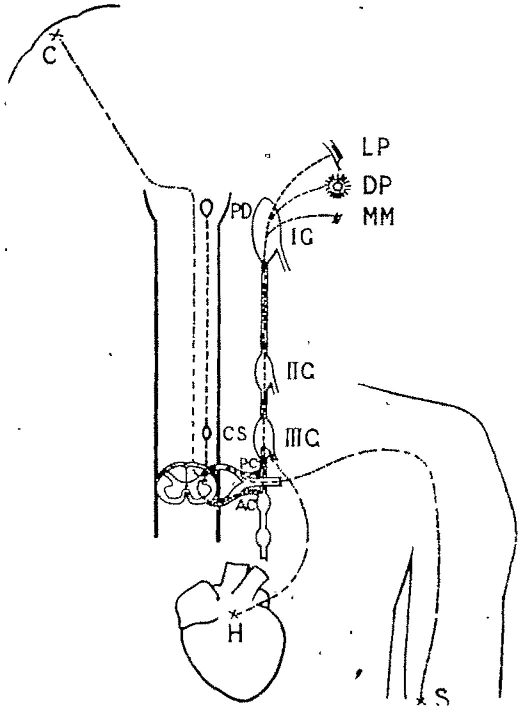


Fig. 10.

Course of impulses in the case. The dots and dashes indicate afferent—the dashes alone efferent tracts.

portion of the levator palpebræ (*LP*). In the construction of this diagram, a figure designed by Purves Stewart[1] has been very useful, and it is a pleasure to acknowledge its assistance. For untiring assistance in unravelling the details of this very interesting case my warm thanks are due to my House Physicians Dr. R. W. Johnstone and Dr. H. A. Stewart.

---

[1] *The Practitioner*, 1905, vol. lxxiv., p. 189.

Since the delivery of this lecture the patient has been almost continuously under observation, and the course of the affection must be mentioned. He was treated by iodide of potassium in ten grain doses three times a day, along with inhalations of amyl nitrite whenever a paroxysm of pain occurred. In the course of a week the severe attacks disappeared, and in less than a month the hyperæsthetic and anæsthetic phenomena were greatly diminished. But at the time of writing this additional note, four months after the patient came under treatment, there are still remains of the motor, electric and trophic changes, as well as some prominence of the left eye, and slight dilatation of the pupil. The hyperæsthesia is still slightly present, but in an almost inappreciable degree. Of the anæsthesia not a trace now remains.

ON A TRIGEMINAL-AURAL REFLEX IN THE RABBIT.

BY SIR VICTOR HORSLEY, F.R.S., F.R.C.S.,

Surgeon to University College Hospital, and the National Hospital for the Paralysed and Epileptic.

IT is well known that if a wild rabbit is feeding quietly its ears lie horizontally along its back, but that when startled it raises itself upright by extending its spine and then erects its ears.

This last movement of the ears has always been regarded as voluntary, *i.e.*, a high purposive act on the part of the animal to detect a source of danger. I have found, however, that such a protective elevation of the ear can also be produced as a simple reflex movement evoked by excitation of only one branch of the trigeminal nerve, namely, the infra-orbital. Of the following photographs, taken at the rate of $\frac{1}{100}$th of a second, the first shows in a young wild rabbit the ears in a position of rest. (I may remark parenthetically that such a little rabbit, even recently caught, will keep quiet for a long time in the warm hand if it is gently stroked, as shown in the photographs).

Photographs 2 and 3, a side and front view respectively, show that when the vibrissæ on the infra-orbital region are gently touched with the finger, or the skin of that part gently pressed (especially over the infra-orbital foramen), the homolateral ear alone is raised up vertically and somewhat rotated forwards.

Photograph 4 shows that the elevation of the ear is tonic and persists for several seconds after the stimulus is removed; the ear then slowly subsiding to the horizontal position.

If the animal was startled and resented the touch of the finger, the contralateral ear was also raised, and the rabbit escaped as shown in Photograph 5, where the hind quarters are shown rising in the action of leaping.

It being obvious that the psychical stimulus of fear caused the bilateral general reaction, I tested other afferent paths when the rabbit was quieted. The results were wholly negative except as regards auditory stimuli. Thus, Photograph 6 shows the ears of the rabbit quite quiet although a ray of direct sunlight is reflected on to its eye; the only result being some protective contraction of the orbicularis oculi muscle. Similarly touch or pressure stimuli applied to the other parts of the body were without effect on the ear.

Pricking of the ears being undoubtedly associated with an appreciation of danger, it is reasonable that it should occur when the infra-orbital region, and especially the vibrissæ, are touched; since a rabbit in escaping from danger encounters numerous obstacles as it runs in the depths of the burrow, or through bushes and grass in the dark, and therefore must largely rely on contact impressions from its face.

Though most active in the wild animal I have obtained this reflex also in tame and albino rabbits, even whether partly lop-eared or no.

Next, as regards the localisation of this reflex movement, the phenomenon is analogous to the representation of the movements of the ear in the inferior colliculis (corpus quadrigeminum posterius), and that the motor path is the same for the auditory and infra-orbital reflexes, inasmuch as the movement executed by the pinna is identical, namely, elevation with rotation so as to present the conchal concavity forwards and outwards.

On the question of the localisation of movements of the ear, Ferrier[1] was the first to find that excitation of the anterior colliculi produced, in addition to movements of the eyes, marked "retraction of the ears," and that the same occurred on stimulation of the posterior colliculi.

V. Bechterew[2] similarly obtained by excitation of both the anterior and posterior colliculi turning of the contralateral ear outwards, in addition to conjugate deviation of

---

[1] "Functions of the Brain," 2nd Ed., 1886, p. 166.

[2] *Archiv. fur Physiologie*, 1884, vol. xxxiii., p. 413.

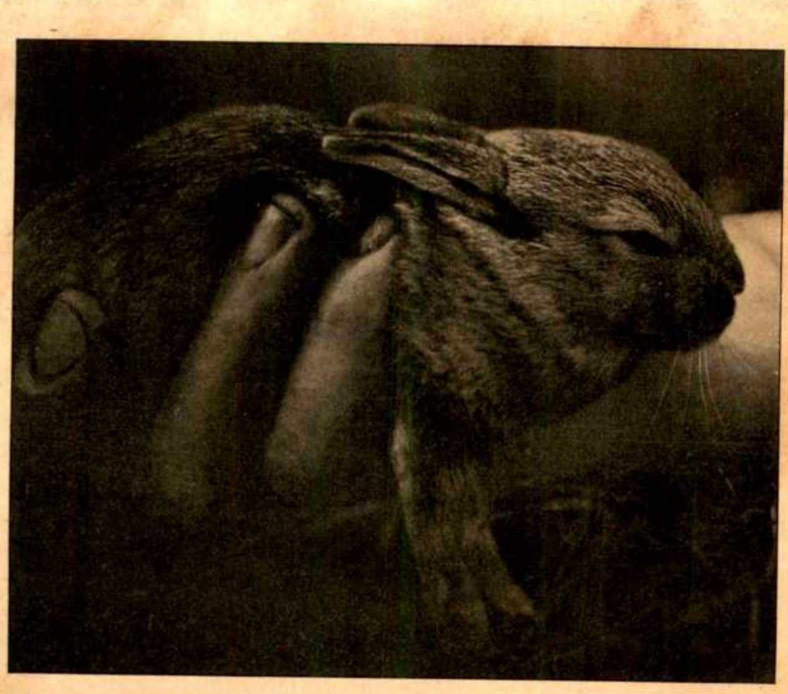


FIG. 1.
Animal at rest; ears horizontal.

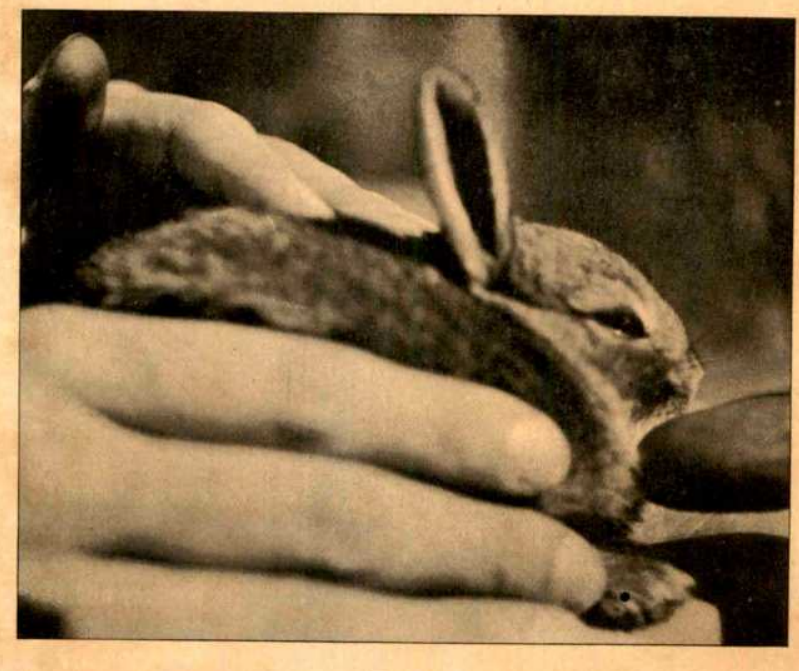


FIG. 2.
Infra-orbital vibrissæ touched with finger-tip; homolateral pinna rising.

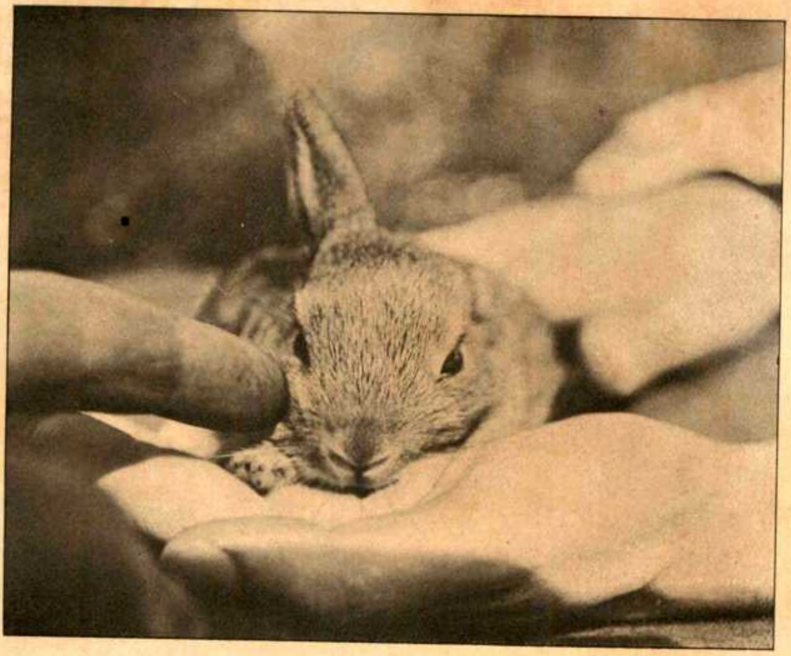


FIG. 3.

Front view of elevation of homolateral pinna only when infra-orbital region touched.

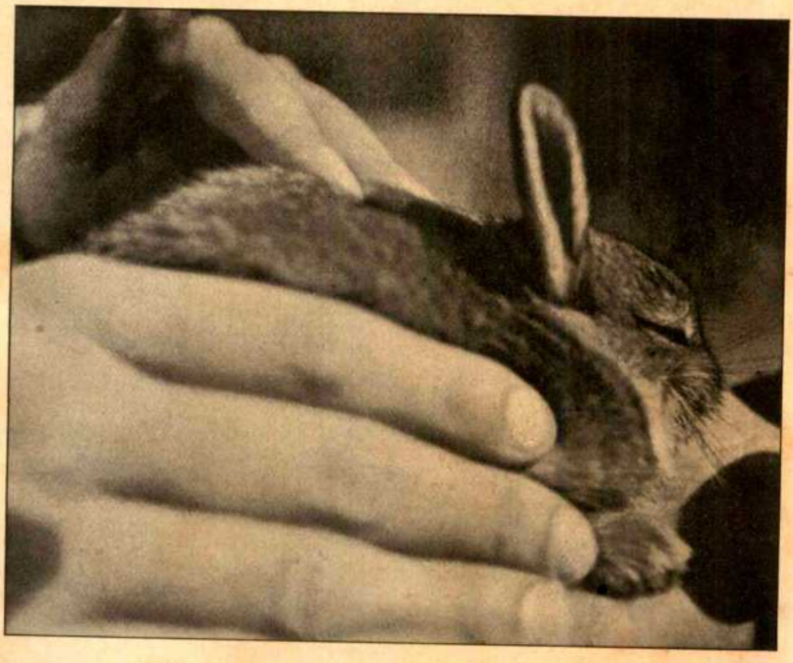


FIG. 4.

Tonic maintenance of the elevation of the pinna for a few seconds after the withdrawal of the stimulation.

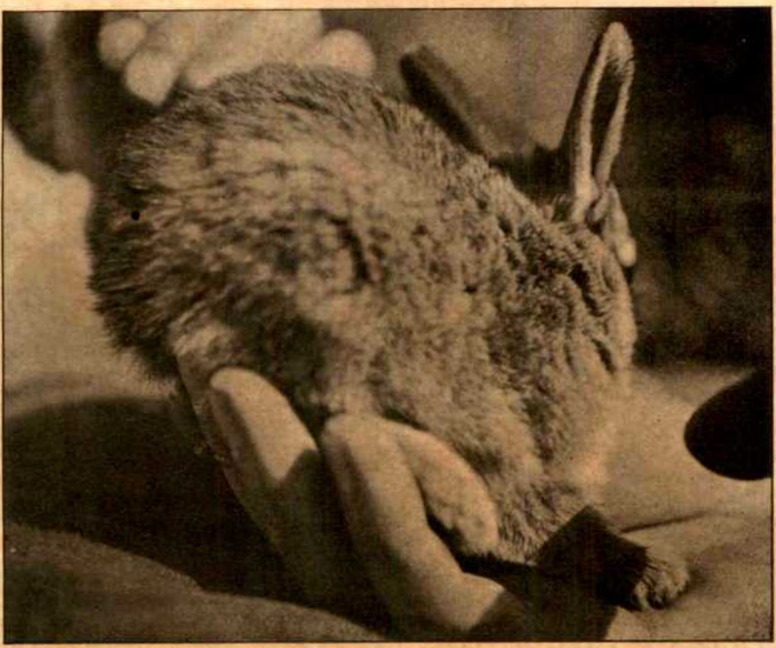


FIG. 5.

Both ears pricked up as the animal is excited and escapes.

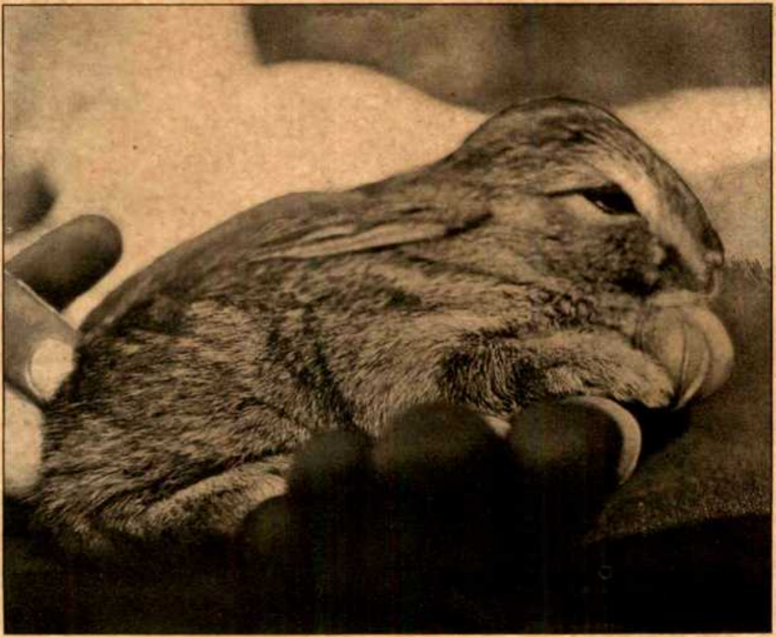


FIG. 6.

Ears horizontal, although ray of direct sunlight is flashed on to eye; example of absence of reaction of the pinna to certain other kinds of stimuli.

the eyes to the opposite side, &c. In a later paper[1] he stated (p. 711) that after unilateral destruction of the posterior colliculus in a rabbit the animal did not prick up the contralateral ear nor turn towards a source of sound.

Prus,[2] in his valuable paper on excitation of the colliculi in dogs, describes the movement of the ear in connection with stimulation of the anterior colliculus only.

Ziehen's[3] observations also coincide with the foregoing results, as also do certain observations by Thiele, which will shortly be published.

During recent years I have made many observations on excitation of the colliculi, anterior and posterior, especially in conjunction with Dr. Magnus during 1904; and in the cat, dog and monkey have observed, in confirmation of Ferrier, that faradic excitation of the outer surface of the posterior colliculus evokes, besides the rotation downwards of the homolateral eye (fourth nerve effect), marked rotation of the pinna of the contralateral ear. The same result was also obtained after section (whether made immediately or three weeks before the excitation) of the intercollicular commissure. The localisation of the phenomenon is therefore exact. Not infrequently the homolateral rather than the opposite ear moves, but the character of the movement is the same, viz., rotation for better audition, an action very different from the flattening and setting back of the pinna as a protection in fighting, which latter can be obtained from a wide area of the cortex cerebri in all animals, notably the ungulata. As, of course, the representation of the cochlear nerve in the posterior colliculus is now definitely established, the similar association with the fifth nerve of the "auditory" movement of the pinna, which evidently is of a defensive and useful character, is easy to understand.

---

[1] *Neurologisches Centralblatt*, 1895, vol. xiv., p. 706.
[2] *Wiener Klinische Wochenschrift*, 1899, No. 45, p. 1,124.
[3] *Archiv. fur Psychiatrie und Nervenheilkunde*, vol. xxi., p. 863.

A NOTE ON THE CONDITION OF THE TENDO ACHILLIS JERK IN DIPHTHERIA.

BY J. D. ROLLESTON, M.A., M.D.OXON.

Assistant Medical Officer at the Grove Fever Hospital of the Metropolitan Asylums Board.

THE condition of the tendo Achillis jerk in diphtheria, though alluded to by a few authors (1), has not received the same attention that has been given to the reflex in the case of sciatica and tabes. The present paper is based on 100 cases of diphtheria in which the tendo Achillis jerk was investigated until the time that the patient was allowed to sit up. This period varied in length according to the severity of the case, the condition of the heart, and the presence or absence of any paralysis, from twenty to sixty-three days from the onset of the disease. The reflex was sought for in the recumbent position. In forty-seven cases the Achillis jerks were affected to a varying degree; they were completely lost in twenty cases, and rendered sluggish in twenty-seven. In the remaining fifty-three cases they remained active throughout the period of observation, which in no case was less than six weeks, and in several cases was prolonged to three months or more.

From this it will be seen that though the Achillis jerks are affected in a large proportion of the cases, this affection is not so frequent as in the case of the knee-jerks. Owing to the greater liability of the diphtheritic toxin to impair the latter, affection of the Achillis jerks was never found without a corresponding or relatively greater impairment of the knee-jerks.

In Hector Mackenzie's (2) cases, the knee-jerks were lost in 80 per cent., *i.e.*, fifty-one out of sixty-four cases. In my own cases they were affected in sixty-nine, lost in thirty-one, sluggish in thirty-eight.

The higher percentage of Mackenzie's figures is probably due to the fact that his cases included a larger number of severe attacks than my own series, and that their course was not modified by serum. Early use of antitoxin, as I have shown elsewhere (3), diminishes the risk of subsequent paralysis. The same probably applies to affection of the reflexes.

As will be seen from Table I., the frequency and degree to which the Achillis jerks were affected bore a direct relation to the character of the initial attack, thus exactly corresponding with the frequency of the most characteristic complications of diphtheria, namely, paralysis and albuminuria (Table II.).

TABLE I.

Classes I. to VI. represent various degrees of severity of the faucial attack.

		CONDITION OF ACHILLIS JERKS.		
	No. of Cases.	Lost.	Sluggish.	Not affected.
Class I.—Very severe	15	9	5*	1†
,, II.—Severe	22	7	5	10
,, III.—Moderately severe	9	2	5	2
,, IV.—Moderate	23	2	5	16
,, V.—Mild	28	0	6	22
,, VI.—Very mild	3	0	1	2

* One died on tenth day, and one on eleventh day. † Died on fourth day.

This table clearly shows that not only is affection of the Achillis jerks commoner in the severe than in the milder forms of the disease, but that in the first two classes in which Achillis jerks were affected at all, the majority were completely lost; the minority were rendered sluggish only. In classes III. and IV. exactly the reverse holds good. In the last two classes the Achillis jerks were not lost in a single instance.

TABLE II.

Showing incidence of paralysis and albuminuria in 100 cases where the Achillis jerks were investigated.

	Paralysis cases.	Severe forms only.		Albuminuric cases.	Persistent albuminuria, i.e., for three weeks or more.
Class I.	13 = 86·6 %	11	= 73·3 %	14 = 93·3 %	6 = 27·2 %
,, II.	9 = 40·9 %	3	= 13·6 %	11 = 50·0 %	0 = 0
,, III.	2 = 22·2 %	0	= 0	4 = 44·4 %	1 = 11·1 %
,, IV.	4 = 14·2 %	1	= 4·3 %	8 = 34·7 %	1 = 6·6 %
,, V.	1 = 3·5 %	0	= 0	4 = 14·2 %	0
,, VI.	0	0		0 = 0	

Further analysis of the cases will show the relation of the affection of the reflexes to paralysis. Thus, out of twenty cases in which the Achillis jerks were lost, in fourteen (70 per cent.) paralysis occurred, and in twelve was severe.

Among twenty-seven instances in which the Achillis jerks were sluggish, there were seventeen cases (62·9 per cent.) of paralysis, of which eight were severe. In those cases where the knee-jerks alone were affected, the incidence of paralysis was much less. Thus, in thirty-one where the knee-jerks were lost, there were seventeen cases of paralysis (54·8 per cent., of which thirteen were severe. Of thirty-eight cases in which the knee-jerks were sluggish without being lost, there were twelve paralysis cases (31·5 per cent.), of which eight were severe.

In all the cases where definite paraplegia was present knee-jerks and Achillis jerks were lost; but in some, where from the severity of the initial attack loss of motor power might have been expected, the patient was able to walk alone from the first, with but slight ataxia and Rombergism, although the knee and Achillis jerks were completely lost.

This abortive condition, to which Aubertin and Babonneix have given the name of "*paraplégie diphtérique fruste,*" is characteristic of the great majority of diphtheritic palsies, as I have illustrated elsewhere (3).

In thirty-two cases of loss of motor power, varying in degree from complete paraplegia to an ataxic gait combined with Rombergism, the Achillis jerks were affected twenty-four times. In thirteen of these they were completely lost; in eleven they were sluggish. The date at which the Achillis jerks were affected varied in different patients.

In the great majority of my patients, viz., thirty-two out of forty-seven, the Achillis jerks were already affected to a certain degree on admission to hospital. In eight of the thirty-two they were completely lost; in twenty-four they were sluggish; 50 per cent. of the knee-jerks were already affected on admission, fifteen being lost, thirty-five being sluggish.

All but four patients, two of which had already reached the eighth, one the fourteenth, and one the thirty-fifth day of disease respectively, were in the first week of their illness, the average time during which they had been ill being 3·92 days. It is interesting to compare with these figures the results of H. Mackenzie, who states that early disappearance of knee-jerks is extremely rare, except in fatal cases. Macdonnell (4), on the other hand, who pointed out that knee-jerks may be lost on the first day of the disease, did not attach any prognostic importance to their absence. As only four of my hundred cases proved fatal, the difference in my results is to be accounted for by the fact that Mackenzie wrote in pre-antitoxin times.

In the great majority of cases the reflexes which were lost did not return, nor did those which became sluggish under observation resume their normal activity during the time they were in hospital.

As a general rule, the severer the case the earlier the Achillis jerks were lost. In most cases, unlike what happens in tabes, they were affected after the knee-jerks. Undue briskness of the Achillis jerks, with or without a similar condition of the knee-jerks, was found in a few cases during the early stages of the disease. Inequality of the reflexes on the two sides was also observed in some cases. This was especially noted where the reflexes which had been lost returned while the case was still under observation. The Achillis jerk, for instance, might return and resume its normal activity on one side for some time before any response could be obtained on the other. In every case loss or impairment of the Achillis jerks outlasted any paralysis that might have been present. A similar condition was observed in the case of the knee-jerks by Berhardt (5), and also by Mackenzie many years ago, and more recently by Aubertin and Babonneix.

SUMMARY.

(1) The tendo Achillis jerks are affected in a considerable proportion of all cases of diphtheria, though less frequently than the knee-jerks.

(2) The frequency and extent to which they are affected

bear, like albuminuria and paralysis, a direct relation to the character of the initial faucial attack.

(3) They are completely abolished in all cases of diphtheritic paraplegia.

(4) Their absence may be the only evidence of loss of motor power in the lower limbs.

(5) Like the knee-jerks, they are liable to be affected at an early stage of the disease, and to remain absent after disappearance of all diphtheritic paralysis, properly so-called.

(6) Like the knee-jerks, again, they may be unequally affected on the two sides, and, like the former, they may be unusually brisk before they become sluggish and finally disappear.

(7) The Achillis jerk, like the knee-jerk, after it has been lost, may reappear on one side before it does so on the other.

REFERENCES.

(1) E. BRAMWELL. "Contribution to the Clinical Significance of the Absence of the Tendo-Achillis jerk," BRAIN, 1901, p. 554.—[AUBERTIN. *Archives générales de Médicine*, 1901, p. 330.—AUBERTIN AND BABONNEIX. *Presse Médicale*, Fev. 8, 1905. Quoted by E. Bramwell, *op. cit.*]
(2) MACKENZIE, H. *St. Thomas's Hospital Reports*, 1890.
(3) ROLLESTON, J. D. *Practitioner*, November and December, 1904.
(4) MACDONNELL. "On the Early Loss of the Knee-jerk in Diphtheria," *Medical News*, October 15, 1887.
(5) BERHARDT. *Virchow's Archiv.*, 1885. Band 99 S. 403.

Reviews.

Traité de Pathologie Mentale, publié sous la directiôn de M. GILBERT BALLET, par MM. D. ANGLADE, F. L. ARNAUD, H. COLIN, E. DUPRÉ, A. DUTIL, J. ROUBINOVITCH, J. SÉGLAS, and CH. VALLON (pp. 1,600, 26 francs. Paris: O. Doin, 1903).

THIS large volume of 1,600 pages with 215 figures interpolated in the text, and six chromo-lithographic plates, is stated in the introduction to be a treatise on mental pathology. The authors hold the view that the mind is not an entity, and that if psychology has long considered it to be so, it is by virtue of an illusion which leads us to attribute to certain phenomena of consciousness an independence which their physiology does not justify: "aujourd'hui il ne peut plus être envisagé que comme une expression synthétique qui désigne l'ensemble des fonctions ressortissant à l écorce du cerveau." Mental pathology is thus closely subordinated to the pathology of the cortex, and necessarily supposes an organic modification, innate or acquired, transitory or lasting of one or several of the constituent elements of the layers of the cortex. Mental pathology not constituting the entire pathology of the cortex, the question is discussed whether it is the pathology of any special region of the cortex. The views of Meynert, Flechsig, and others, are briefly discussed with the conclusion, "il est vraisemblable que la plus grande partie de l'écorce et peut-être toute l'écorce concourt, dans une certaine mesure, à l'exercice de l'intelligence: au moins actuellement on n'est pas en droit de dire qu'il n'en est pas ainsi." What is affirmed is that integrity of the cortex is the condition of integrity of the intelligence, and reciprocally that mental disorders presuppose a perturbation of one or several of the conditions regulating the normal functions of the cortex.

The discovery of the lesions of general paralysis, cellular changes which appear to condition certain deliria of infectious or toxic origin, and the relation of mental troubles to various toxæmias have lent new support to the doctrine which subordinates disorders of the intelligence to material lesions or disorders of nutrition of the cortical cells.

With regard to classification, the authors remark that an imperfect science is only susceptible of imperfect classification, which is the case in mental pathology. Certain conditions, such as mania and *confusion mentale*, are syndromes of varying etiology and, therefore, cannot be classed in the same rank and on the same plan as affections constituting perfectly defined nosological individualities such as intermittent insanity or progressive general paralysis.

The work is divided into ten books. The first is concerned with the history, general etiology, diagnosis, and semeiology of mental disorders, and, for the historical section, Dr. Ballet is responsible.

The second chapter of the first book on the etiology of mental affections is by Dr. Anglade, who gives a comprehensive table dividing causes into: (I.) Le terrain psychopathique, and (II.) Les causes déterminantes des psychopathies.

The latter are subdivided into:—

(*a*) Biological and physiological (age and sex).

(*b*) Social, political, religious, intellectual and moral.

(*c*) Mechanical (concussion, trauma, compression, &c.).

(*d*) Physical (cold, heat, light).

(*e*) Pathological (affections of nervous system, of other organs, infections, and intoxications).

With regard to the influence of heredity in the causation of insanity, a brief summary is given of the conflicting views of Weissulann and others, as to the transmission of acquired peculiarities, and the opinion is held that what is transmitted, anatomically speaking, is an imperfect development of one or several central "territories," but with the saving clause that it is best to avow that we know nothing about the organic conditions of the *terrain psychopathologique*. Reference is made with disapproval to the attempt to differentiate a special group of cases to be known as *folies héréditaires*, as the term could not be applied specially and exclusively to certain forms of mental disorder.

The chapter on etiology occupies forty-seven pages, a summary being given of the present state of knowledge as to the causes of insanity, with many references to recent literature.

With regard to the influence of uterine and ovarian disease in the production of insanity in women, Dr. Anglade says: "Chez la femme, les lésions de l'utérus et de ses annexes ont été souvent incriminés pour expliquer la folie et on leur a fait jouer un rôle qu'on n'est véritablement pas en droit de leur attribuer."

Under the head of infections reference is made to the as yet inconclusive evidence of a microbic origin of "acute delirium," and to the effects of influenza, typhoid fever and malaria. He argues with Clouston as to the special liability of tubercular patients to suffer from suspicion and ideas of persecution, although other forms of mental disorder may occur in this connection. The importance of auto-intoxication is fully referred to with the statement that the nature of the poison is at present problematical.

Chapters III. and IV. of Book I. are by Dr. Séglas on the methods of examination of patients and the semeiology of mental diseases. The subject of measurement of the skull in various directions, and the calculation of cranial and facial indices is discussed at length, but the limitation of the value of these measurements in psychiatry is recognised. Five pages are devoted to deformities of the ears, which are not considered to be notably more frequent in the insane than the sane.

The present state of knowledge as to such questions as auto-intoxication, urotoxicity, and bacteriological examination of the blood, are fully considered.

The psychical symptoms of insanity are studied under the headings of (1) disorders of language (spoken and written); (2) the acts of the insane; (3) morbid states of cœnesthesis; (4) states of intellectual enfeeblement and debility; (5) hallucinations; (6) delirious ideas (= Wahnideen and delusions).

Mercier's classification of delusions is given in full with approval.

Book II. contains two chapters only on mania and melancholia by Anglade and Ballet.

These conditions are described as "syndromes" of variable etiology and uncertain pathology, and cannot, therefore, be looked upon as definite diseases. It is worthy of note, however, that the authors do not relegate these time-honoured names to the past, but recognise that they still have a considerable clinical importance.

Book III. is connected with mental disorders in intoxications and infections. With the exception of the chapters on délire aigu and confusion mentale, which are written by Dr. Anglade, the whole of this section is from the pen of Dr. Roubinovitch. Psychical poisons are divided into exogenous (organic and inorganic), endogenous (diabetic, uræmic, hepatic, and thyroidal), and bacterial (infections.)

The chapter on alcoholism and the alcoholic mental disorders is very complete, and many diagrams, illustrating the relationship of alcoholism and crime in different countries, are given. Great stress is laid on the "prophylaxis" of alcoholism by legislation and general education.

Separate chapters are devoted to délires fébriles, délires des septicémies, and délire du collapsus.

Dr. Roubinovitch limits the latter to mental disorders supervening in cases of sudden adynamia accompanying certain grave febrile affections, and differs in this respect from Kraepelin's description of Collaps-delirium.

Dr. Anglade devotes two chapters to délire aigu and confusion mentale. He considers the former equivalent to the delirium acutum of German and acute delirious mania of English writers, and that it is not merely a very acute form of mania nor, on the other hand, a febrile delirium. The observations of Bianchi and others as to a specific micro-organism in the blood in this condition are mentioned with the avowal that French alienists show little partiality for this view of its origin. Dr. Anglade, however, considers it to be of "nature infectieuse," as shown, not only by its symptomatology, but by the diffuse visceral lesions it produces.

"Confusion mentale" is defined as a syndrome which ordinarily develops primarily as the result of intoxications and infections, but which may also secondarily complicate other mental affections (mania, epilepsy, alcoholic delirium, and organic cerebral affections).

The following synonyms are given :—

Démence aiguë (Esquirol) ; stupidité, stupeur (Georget, Dagonet) ; confusion hallucinatoire (Delasiauve) ; torpeur cérébrale (Ball); hallucinatorischer Wahnsinn (Krafft-Ebing); amentia (Meynert, Serbsky) ; mania hallucinatoria (Mendel) ; acute confusional insanity (Conolly, Norman) ; paranoia dissociativa (Ziehen).

Dr. Anglade considers "confusion mentale" to be neither a pure vesania nor a simple state of intoxication or exhaustion, but a combination of psycho-neurosis and cerebral infection. He groups many post-operation, puerperal and post-febrile cases under this group, and describes two principal forms : (1) stupidité, or confusion mentale asthenique, and (2) délire hallucinatoire aigu, or confusion mentale délirante hallucinatoire.

Book IV., on "psychoses constitutionelles," is entirely written by Dr. Arnaud, and consists of three parts :—

(1) Psychoses chez les sujets à prédisposition latente.

(2) Psychoses chez les sujets à prédisposition apparente.

(3) Démence vésanique.

Part I. contains as its principal subject a description of "délires systematisés ou partiels," the equivalent of Paranoia or Verrücktheit of German writers. A most comprehensive summary is given of the views of writers in different countries as to this much-debated subject, and a most valuable table of German classification of Paranoia and French equivalents. Dr. Arnaud admits of an acute and curable, as well as a chronic and incurable, group of cases of this nature. The whole subject is treated at great length and with complete thoroughness.

The second chapter of this part is on "psychoses périodiques ou intermittentes," and is well illustrated by charts of the course of such cases.

Part II. contains the consideration of manie raisonnante, folie morale, persécutés-persécuteurs, délire systématisé originel, obsessions (including sexual anomalies and perversions), exaltation maniaque et mélancholie raisonnante. The author abandons the term "dégénérés" for individuals of this group, and prefers the expression "déséquilibrés" as not implying any doctrinal notion.

Part III. discusses démence vésanique tardive and démence vésanique précoce. The question of "dementia præcox" is fully considered. With regard to Kraepelin's inclusion of paranoid forms in dementia præcox, Dr. Arnaud says: "Nous continuons à penser avec tous les observateurs français que la démence précoce et les délires systématisés dans leur ensemble doivent être séparés dans les classifications aussi nettement qu'ils se distinguent en clinique, et que la dénomination de *démence paranoïde* doit être limitée à certain cas de démence précoce qui débutent par une phase de délire vaguement systématisé."

Book V. treats of the neuroses considered only from the point of view of the mental disorders occurring in them, the first chapter on the "Etat mentale des hystériques," being written by Dr. Colin, and the remainder on mental disorders in epilepsy, psychical epilepsy, chorea (both Sydenham's and Huntington's chorea), and Parkinson's disease by Dr. Dutil.

Dr. Colin describes the short delirious manifestations of ordinary hysteria, as well as somnambulism and ambulatory automatism. He does not believe in the existence of a special "hysterical insanity" or "hysterical mania," but merely in hysteria associated with insanity of any clinical variety.

The psychical manifestations of epilepsy are described by Dr. Dutil under the following headings :—

(1) Les syndromes avec excitation.
(2) Les syndromes avec depression.
(3) Les formes alternantes, and (4) epileptic dementia.

Dr. Dutil does not admit the existence of a special "choreic insanity," but agrees with Jeffrey that insanity associated with chorea is a manifestation of degeneration.

Book VI. is perhaps the most important in this work, and is entirely written by Dr. Dupré. It is entitled "Psychopathics organiques," and contains chapters on progressive general paralysis, organic encephalopathies (hæmorrhage, softening, meningitis, abscess, tumour, scleroses, syringomyelia, myopathies, &c.), and organic dementia.

The chapter on general paralysis is very complete and profusely illustrated, both in the clinical and pathological sections. The author agrees with Sérieux and Mignot that a "sensorial,' variety of general paralysis exists, characterised by the predominence of aphasia, hallucinations, hemianopsia, &c., and in which the lesions predominate in the middle and posterior part of the cortex. He believes that in certain cases true general paralysis may end in recovery, and considers it illogical to regard these as instances of pseudo-general paralysis.

Under the head of diagnosis he lays great stress on the value of lumbar puncture, the discovery in the fluid thus withdrawn of small lymphocytes staining deeply being almost universal in cases of general paralysis and preceding any motor disorders.

The presence of lymphocytes, however, merely settles the question of the organic or inorganic nature of the case as the same condition is met with in tabes, chronic or subacute meningomyelitis, syphilitic or not, tubercular meningitis, and chronic alcoholic meningitis. He sums up as follows: "Le résultat positif du cyto-diagnostic cephalorachidien n'indique l'existence de la paralysie générale que lorsqu'il n'existe aucune raison, étiologique ou clinique, d'imputer à un autre processus méningitique la lymphocytose constatée."

A very full description is given of Korsakoff's polyneuritic psychosis, which is considered to be an attenuated expression while délire aigu is an intense expression of intoxication of the nervous system in the predisposed. The pathological condition is very fully considered with the conclusion that, "l'affection psychopolyneuritique apparaît comme consécutive à l'imprégnation de l'ensemble du systême nerveux par les poisons en circulation dans l'organisme malade." Under brain tumours, a table is given of the relative frequency of psychical disorders in association with tumours of different areas of the brain, the highest proportion being shown in tumours of the corpus callosum, and next of the frontal region.

Book VII., by Roubinovitch, treats of mental disorders in congenital or infantile cerebral lesions (idiocy and imbecility), and is as complete and well illustrated as the rest of this work.

Dr. Roubinovitch is also responsible for Chapter I. of Book VIII., treating of myxœdema and cretinism, while Dr. Dutil discourses on mental disorders from over-function of the thyroid body (exophthalmic goitre).

Dr. Anglade writes fully in Book IX. of the treatment of mental disorders, and Book X., which completes the work, is by Dr. Vallon on administration, lunacy law, asylums, experts, civil capacity, responsibilities, crimes, life insurance, traumas, feigned insanity, and deontology.

There appears to be only one adverse criticism to make on this monumental work, which is of encyclopædic importance, and redounds in the highest degree to the credit of our French colleagues, and must ever remain a most valuable work of reference.

The criticism is directed against the index, which is merely a table of contents of the different chapters. For such a large work a full index should have been given so as to facilitate the labour of searching through the different chapters for any special subject to which reference is desired. Considering the size and weight of the book, it would have been better in two volumes, but its extraordinarily low price (26 fr.) is an inducement to everyone engaged in psychiatry to purchase it.

R. PERCY SMITH.

Myoclonus Multiplex. By CHARLES L. DANA. (*Journal of Nervous and Mental Disease*, August, 1903).

Pathology of Paramyoclonus Multiplex (Friedreichs Type). By J. RAMSAY HUNT. (*Journal of Nervous and Mental Disease*, July, 1903).

Die Progressive Myoklonus-Epilepsia (*Unverricht's Myoklonie*). By Dr. HERMANN LUNDBORG.

THE above recent monographs on myoclonus may well be reviewed together, for they raise the important question as to what is meant by the name "myoclonus." Is "myoclonus" a disease—an entity—or is it only a symptom? As originally used by Friedreich, it certainly indicates a disease having definite clinical symptoms. Unverricht attached the name to a class of cases differing entirely from that described by Friedreich. How, then, should the term "myoclonus" be used?

Dana starts by the statement that myoclonus is a general term covering all the diseases in which muscle-twitching is a prominent symptom. The term here is used as indicating a symptom, and as such he proceeds to classify the diseases in which the symptom occurs; the classification he arrives at is as follows:—

(1) Paramyoclonus multiplex, of Friedreich; astasic myoclonia, of Vanlair; multiple spinal myoclonus, of Lowenfeld; fibrillary chorea, of Morvan; fibrillary myoclonus, of Kny.

(2) Functional or hysterical myoclonus multiplex, chorea major, chorea electrica, of Henoch.

(3) Myospasmia; memory-spasms, of Friedreich; habit chorea; chorea variable des degenerès; convulsive or spasmodic tic; tic general; Tourette's disease, myriadrit, palmus; tic neurosis, of Collins.

(4) Degenerative chorea, hereditary chorea, Huntingdon's chorea; myoclonus-epilepsy; myoclonus, of Unverricht, of "familial" type; myoclonia congenita, of Seeligmüller (?); hereditary degenerative chorea of Sachs.

(5) Infectious chorea, chorea minor, Sydenham's, chorea; chorea electrica, of Dubini (?); chorea electrica, of Bergeron; senile chorea, of Gowers (?).

It is difficult to see what advantage can be derived from a classification which puts under one head diseases which are essentially different in type.

The "tics" form a most useful clinical group, and, as a type, are easily distinguished from Sydenham's chorea. If the term "myoclonus" is to include all diseases in which spasmodic muscular movements occur, which neither clinically nor pathologically—so far as pathology goes—have anything in common, then the term loses its significance and has but little value.

It would be well that the term "paramyoclonus multiplex" or "myoclonus multiplex" should be reserved for that form of myospasms characterised by multiple isolated contraction of individual muscles.

Hunt goes still further, and would distinguish contractions of this type from the contractions of a cerebral type which are characterised by movements of a more or less co-ordinate nature.

It would be well if the term "myoclonus" could be reserved for the two conditions above mentioned, viz., the paramyoclonus of Friedreich and the myoclonus of Unverricht.

Hunt has had the opportunity of investigating pathologically one case of myoclonus of Friedreich type. He finds no abnormality of the nervous system, except some slight change in the vessels of the gray matter of the spinal cord. The muscles, on transverse section, have a diameter of unusual size, measuring in some instances 175 micromillimetres. In contrast to this are very small fibres having crescentic forms and flattened out on the periphery of the large fibres. Instead of finding the nuclei of the sarcolemma sheath confined to the periphery of the muscle fibre and just beneath this membrane, they are found scattered as well between the sarcous elements. Sometimes three or four nuclei occupied a central position in a single fibre. The nuclei of the sarcolemma sheath are increased in number.

Lundborg, in an excellent monograph on *Die Progressive Myoklonus-Epilepsia*, divides the myoclonias as follows:—

(1) Myoklonia symptomatica.

(2) Myoklonia simplex, or myoklonus multiplex.

(3) Myoklonus-epilepsia, divided into two forms: (*a*) a progressive form (Unverricht's family type); (*b*) an intermittent sporadic form.

The tics, Huntingdon's chorea, should not be confused with the myoclonia, from which they are easily distinguished.

This classification would commend itself to the writer of this review, not only on account of its simplicity, but also because it allows of a wide use of the term as a symptom in association with other diseases, and recognises two well-marked types of disease which may be considered as entities.

The monograph deals more especially with the one form of myoclonus epilepsy, and the following description of the disease is given:—

The disease begins in later childhood and lasts during life, so that some of the patients between 60 and 70 years old have suffered from fifty to sixty years.

Most patients, however, die about middle age. Three stages are described: the first, characterised as the epileptic-tetaniform stage, is of comparatively short duration. The attacks occur mostly at night and during sleep, at first with a long interval, but always tending to become more frequent.

The patient awakes out of sleep with painful tonic or clonic and tonic cramp in various parts of the body. The attacks are very different in their intensity. In the mildest attacks rapid muscular contraction takes place in the extremities. In the severest cases the attack resembles tetanus, and the contractions follow one another so quickly that the muscles never become flaccid. These contractions do not produce a locomotor effect, owing to the fact that the antagonistic muscles are in like contraction. Every manipulation tends to increase the spasm. Consciousness is preserved, and the patient has pain and distress, and may cry out with pain. Urine and fæces are not passed incontinently.

The second stage, so-called myoclonic epileptiform stage, lasts for several years (ten or more). The disease is now more pronounced during the day. Tremor — fibrillary and fascicular muscular contractions occur — beginning usually in the upper extremities, then in the buttock muscles, the throat, the face, and, lastly, the diaphragm, the pharynx and larynx, and, finally, the ocular muscles and sphincters of the bladder and rectus—in a word, all the voluntary muscles.

The patient experiences good and bad days.

During this stage psychical phenomena present themselves and the intelligence falls off; the patients exhibit changes between depression and exaltation.

The third—terminal—stage is one of cachexia. This may begin at almost any time, but generally shows itself after the first decade of the disease.

The muscular movements gradually increase in the course of years, whilst the epileptic attacks become less frequent, and eventually disappear. The periodicity of the attack becomes irregular, and the intermediate periods of rest shorter.

Rigidity of muscles now manifests itself, and the patient assumes a fixed and bent position. Any attempt to alter the position produces a definite resistance. The patient sweats and salivates profusely, passes into a somnolent half-stupor stage. Urine and fæces are often passed incontinently; the cachexia increases, and the patient dies of inanition or of some complication.

Two factors in the etiology of the disease came into prominence; the first of these is the hereditary and family nature of the disease, and secondly the influence of alcohol.

In two families there were five cases; in one family four cases; in seven families three cases; and in nine families two cases.

The occurrence of paralysis agitans in other members of the same family is a striking feature brought out in the cases recorded, for in one family in which five cases of myoclonic epilepsy occurred five members of the family were affected with paralysis agitans.

The author next deals with the differential diagnosis of the disease.

The important diagnostic points are:—

It is a family disease—starting with nocturnal epilepsy—associated in childhood with attacks resembling tetanus, and after a few years lightning-like involuntary muscular contractions which gradually affect all voluntary muscles. The patient has good and bad days. The muscular contraction frequently affects only a part of a muscle. The contractions occur irregularly and are increased by voluntary movement; during sleep they stop or are very much diminished. As time goes on a definite muscular rigidity occurs. The knee-jerks are exaggerated. No trophic disturbance of muscles or abnormality of sensation is present. Sweating and salivation are marked features. After some years failure of mental powers, sometimes associated with mania and hallucinations occur.

The author next contrasts the disease with chronic progressive chorea (Huntingdon's chorea). Both diseases arise from a degenerative basis, have a progressive course, and end in dementia. The involuntary contraction of muscles is present in both cases.

The points of distinction are:—

In Progressive Myoclonus Epilepsy.	*Huntingdon's Chorea.*
(1) Direct heredity unusual.	(1) Direct heredity usual.
(2) Disease begins in childhood, with nocturnal epilepsy—and later on the muscular contractions appear—the muscular contraction and the attacks stand in relation to one another.	(2) The disease affects persons between 30 to 40 years of age and is rarely combined with epilepsy.
(3) Periodicity dependent on various sources of reflex irritation.	(3) No periodicity.
(4) Sensory clonic reaction present.	(4) Sensory clonic reaction absent.
(5) The muscle contractions are myoclonic— Sharp contractions affecting single muscles.	(5) The muscle contractions are choreic— Slow contractions affecting groups of muscles.
(6) On intention movement the contractions increase.	(6) On intention movement the chorea becomes less.
(7) The patient soon loses the power of walking.	(7) The power of walking, though difficult, is not lost till the final stage of the disease.

The differential diagnosis between this disease and Koschewnikow's epilepsy and Dubini's chorea electrica is then discussed, and the similarity of myotonia (Thomsen's disease) to myoclonia is pointed out.

PATHOLOGICAL ANATOMY.

In the few cases which have been examined, the changes found have been slight and often attributable to some intercurrent condition.

In Clark and Prout's cases changes were found in the second layer of cells of the cortex, and also in the large pyramidal cells of the third layer—and these authors suggest that these changes underlie the myoclonic spasm.

It would seem, therefore, probable that myoclonus is dependent on changes in the Rolandic region of the cortex.

The possible relation of the disease to changes in the thyroid and parathyroid is discussed, for it has been shown experimentally that clonic spasms occur after removal of the thyroid.

Ramsay Hunt, in the examination of a case of paramyoclonus multiplex, which would come under Lundborg's second division, could find no change in the nervous system, but found changes in the muscles, the fibres being of an unusually large size—with an increase of nuclei in the muscle fibre and also in the sheath of the muscles.

In conclusion of this short review, it would seem that but little advantage is gained by placing under the term myoclonus all the various conditions enumerated by Dana, and it would be far better to adopt the classification given by Lundborg:—

(1) Myoklonia symptomatica — using the term myoclonus simply as a symptom indicating a form of myospasm characterised by multiple spontaneous, and isolated contraction of individual muscles.

(2) Myoklonus multiplex.

(3) Myoclonus epilepsy—(*a*) A progressive form. (*b*) An intermittent form.

FREDERICK E. BATTEN.

Isolement et Psychothérapie. Traitement de l'Hystérie et de la Neurasthénic. Pratique de la ré-éducation Morale et Physique. By Drs. JEAN CAMUS et PHILIPPE PAGNIEZ. Préface du Professeur J. DEJERINE. (Paris: Félix Alcan. 1904. Pp. 407.)

THE object of this book is to demonstrate the method and the results that accrue in the treatment of hysteria and neurasthenia, as it is now carried out at the Saltpêtrière in the wards of Prof. Dejerine. In a short but interesting preface Prof. Dejerine remarks on the neglect of moral force by medical men in their treatment of nervous diseases; he considers that although moral influence is at last beginning to be more adequately recognised as a power in therapeutics, it is still far from sufficiently appreciated. Finding that the results obtained by the use of the various factors that make up the Weir-Mitchell treatment did not come up to his expectations, he was gradually led to see that this treatment should be regarded only as a means to bring the patient under moral influence, that is, in the current phraseology, as a means to the use of psychotherapeutics. Further, the inconveniences, and often bad results that arise from the treatment of hysterical patients in a general ward of a hospital and from the effect upon them of the presence of other patients similarly affected, led him to devise a plan by which poor patients in hospital could have the same advantages as the rich in private nursing homes. This he accomplishes by isolating each bed by means of curtains, and since he has adopted this measure the results of hospital treatment have been as good as those obtained in nursing homes in patients of the well-to-do classes.

The authors have carried out Prof. Dejerine's mode of treatment under his supervision, and are thoroughly conversant with it. In this book they attempt the somewhat difficult task of describing all the details of the method. Obviously where so much depends on the individuality of the physician, and on the idiosyncrasies of each patient, and where the treatment rests upon such an undefined and intangible quantity, as the moral influence of the one upon the other, any description which can present all the points to a reader without obscurity is a matter of great difficulty. Again, the very secret of success eludes description, because it lies in the physician's ability to seize upon the higher moral qualities of his patient, and to enlist them into his service to overcome the lower tendencies which have resulted in nervous disorder. To their general account of the method, the authors have, therefore, wisely added the description in detail of a number of cases. These cases are well reported, and each ends with some short critical remarks on the character of the illness, and the result of the treatment. More than one-third of the book is taken up by these case reports. The first part of the book deals historically with the subjects of isolation, of "faith cures" by various means, of hypnotism and suggestion, and of psychotherapy by "persuasion." These several topics are treated in the widest possible sense, and the historical *résumé* given cannot fail to be of interest. Under isolation the authors discuss hermits, religious retreats, life in monasteries and convents, and allude to a now-forgotten but former popular author, Zimmermann, of whom O. W. Holmes said that he was the only man to whom he would intrust his latch-key.. "Suggestion" is carefully distinguished in this book, as in that of Prof. Dubois (of Berne), from "persuasion"; the former is condemned, and is to be superseded by the latter. The distinction though clear in theory, in practice would seem to become rather fine, especially from "suggestion in the waking state." Under the history of persuasion there is included a short but interesting account of that form employed by the Roman Church as "spiritual direction." The second part of the book is that which embodies the essential teaching of the authors. In it they discuss fully the best means of carrying out isolation of nervous patients under varying circumstances, the rules for rest and dietetics, and for the employment of massage and electricity.

A chapter on the influence of the mind on the body, and on the reverse effects of the body on the mental state, leads to a discussion on hypnotism, which is rejected as a therapeutic agent, partly on account of the difficulties and dangers inseparable from its employment, partly because other less objectionable measures can be substituted for it with good results, the method of persuasion being, as said above, employed instead of suggestion.

The manner of carrying out this method by the physician is discussed in full detail, and instructions are even given as to what he should do and say during "entretiens psychothérapiques"; a considerable part of psychotherapy lies in successfully impressing upon the patient that he or she is suffering from a curable affection, and, afterwards, "persuading" them of the improvement that is taking place in their condition. The authors, however do not tell us what we should do with that very troublesome person with whom we sometimes meet, who is never tired of insisting that he is no better, nor with that neurasthenic who is not disposed to take anything on trust, but is prepared to argue about treatment with his doctor to an unlimited extent. It further seems to follow that successful "psychotherapy" almost demands as a *sine quâ non* that the doctor should be the intellectual superior of his patient. This is not always the case.

A passing allusion to a useful chapter on the re-education of hysterical and neurasthenic patients, that is the restoration of their physical, moral, and intellectual powers to the normal standard of each, that they may again take their places as normal members of society, and of the means to be employed in this difficult task, may conclude this notice of a book which is well written, highly interesting, and deals with the beginnings of a rational mode of treatment which is likely to proceed to further development in the future.

J. Michell Clarke.

On Unilateral Hallucinations of Hearing. By E. Lugaro (Sulle Allucinazioni Unilaterali di Audito). *Rivista di Patologica Nervosa e Mentale,* May, 1904.

Dr. Lugaro reports a case of hallucinations of hearing assigned to one ear. The patient was 39 years old, a smith by trade, had an aunt and one sister insane. In February, 1898, after the death of his father, he fell into a deep melancholy. He tried to kill himself with a small revolver, the charge of which burst the tympanum and lodged in the temporal bone. Most of the contents were extracted; probably some fragments remained, for suppuration continued. He was sent to the asylum, where he improved so quickly that he was discharged after twenty-four days, committed to the charge of his wife. He returned to his work. Two years and six months after, his wife meeting with an accident, he talked of suicide, became maniacal, and was again brought to the asylum. He was motionless, silent, and passive, refused food, and did not sleep. He said that the voice of his dead father was ever in his right ear, repeating "suicidati," but he resisted the suggestion. He said that for two years back there was a continuous noise in his ear, like the cry of a cricket, but low and uniform. In a month he was well again and able to leave the asylum. The sound in the ear continued, but did not take the form of words. After a time the hallucinations of hearing returned. Then he began to hear what he had been thinking repeated into his right ear. In the course of two years the hallucinations became more frequent, returning almost every day. He fell into a state of melancholia attonita, and was again brought to the asylum. He was thin and anæmic; the right tympanum was found to be loose, but cicatrised. Hearing gone on the right side, but there was unusual excitability to the galvanic current. The patient was soon again able to go home and return to his usual work. Dr. Lugaro can only find two other examples of the hallucinations of voice repeating the thoughts. In this case we have a peripheral irritation and a morbid state of the brain. Unilateral auditory hallucinations, however, have been described by many observers. In some cases it looks as if the affection of the ear was the cause of the insanity, though we may assume a morbidly excitable condition of the brain. The peripheral lesion favours the production of hallucinations though not a sufficient cause, without an abnormal activity of the brain. Mabille has described a case in which the hallucinations ceased after the ear disease had been cured. Lugaro tells us that all the insane who are deaf are affected with auditory hallucinations. Lunatics who are blind may still have visual hallucinations.

William W. Ireland.

Die Färbetechnik für das Nervensystem. By Bernhard Pollack. (Third Edition. Berlin, 1905. S. Karger. Price 3s. 6d. net.)

Dr. Pollack's compendium is arranged as follows: (1) The removal of the brain, its naked-eye examination, and its preparation for museum purposes; (2) hardening fluids and microscopic technique; (3) the influence of preservatives on brain weight, micrography and photography (a chapter of three pages only); (4) methods of staining the three chief constituents of nervous tissue; (5) general remarks.

In his chapter on methods of hardening, the author emphasises the axiom, the better the fixation the better the microscopic specimen. The postulates of an ideal fixative are these: it shall fix without shrinking; it shall serve as an imbedding medium, and so do away with the necessity for dehydration and clarification; it shall act rapidly, but not cause brittleness when immersion is prolonged, and it shall be neutral in the presence of different stains, so that sections from one block may be variously treated and display all constituents. But, of course, we are still far off our ideal. Pyridin, which, by the way, does not occur in Dr. Pollack's list, comes nearest, for it combines the properties of a fixer, a clarifier, and a dehydrator, but it has disadvantages in causing shrinkage and in requiring paraffin imbedding, not to mention its offensive odour. In permitting the use of many methods, formalin almost satisfies one postulate, and it rightly occupies the place of honour in this compendium.

In the series of staining procedures we observe none that has not been described elsewhere, but the running commentary—an important feature of the work throughout—is full of sound suggestions, and proves the writer to be a thoughtful, accomplished, and well-practised microscopist. After Weigert, it is insisted that a perfect stain should be selective, certain, time-saving, intense in colouring, and permanent; that, for the sake of orientation, it should make other objects in the field visible, and that it should not require drastic, intercurrent treatment.

We know too well that it is impossible for any worker single-handed to test the merits of the ever-increasing multitude of neuro-histological methods; therefore, rather as co-operators than as critics, we will indicate some omissions and shortcomings which we have noticed, and which, we hope, will receive attention in future editions. The method of Marchi in its original form is obsolete; as workers at Queen's Square have demonstrated, when it is desired to follow a degenerated tract of nerve fibres, hardening in formalin and the use of Busch's osmic acid and iodate of sodium mixture are much more advantageous; while, if the tissue be hardened in Müller's fluid, we follow Dr. Orr, and assist the osmic penetration with acetic acid. Again, if we wish to display normal nerve fibres in formalin-hardened tissues, we find in the solutions of fluorchrome and of chrome alum, used by the late Professor Weigert for other purposes, excellent mordants for myelin. The mixture of perchloride of mercury and picric acid which finds favour as a fixative among Italian histologists, Unna's polychrome blue as a stain for nerve cells, the platinum method of Dr. Ford Robertson for displaying neuroglia, and the fresh method of Dr. Bevan Lewis, all deserve notice; and, finally, the remarks on micrography should be either extended or cut out.

Apart from these imperfections, the compendium will prove a useful companion to the more comprehensive work of Bolles Lee. The author's discrimination between important and unimportant methods is called for, and we welcome the complete list of references appended to each chapter.

ALFRED W. CAMPBELL.

The Anatomy of the Brain. (*A Study of the Human Brain from the Brain of the Sheep.*) By J. F. BURKHOLDER, M.D., Professor of Anatomy in the Illinois Medical College. With an introduction by Professor HENRY H. DONALDSON, of Chicago. (17[illegible] pages, octavo, 32 full page plates [5 coloured]. Cloth. Price, 2 dols. post paid. Chicago: G. P. Engelhard & Co.).

AT times when human material was unprocurable for teaching purposes, Professor Burkholder tested and proved the usefulness of the brain of the sheep, and he now issues a guide to its dissection.

The manual will be welcomed by the student of medicine, and even more so by the graduate, for it will enable them, independently and privately, to acquire familiarity with, and renovate, their knowledge of an anatomical "part" as difficult as it is important.

The various dissections are carefully planned, the illustrations well reproduced, and, the descriptions, on the whole, correct and commendably brief; but the attention of the reader is too frequently arrested by confusion of tenses, by loose construction, and by misprints, all of which should have been eliminated before the "revise" was approved. It is also to be regretted that the author has intentionally omitted references to homologies, for much that was previously obscure in this direction has been removed by the recent researches of Elliot Smith, and others, and surely the interest of the student would be accentuated if he were told the human equivalents of the various gyri and sulci coming under inspection.

The English tongue may trip over the expression "hemisphærium cerebri," still it is pleasing to find Professor Burkholder adopting the nomenclature recommended by the Basle convention.

ALFRED W. CAMPBELL.

Essai sur les Alterations du Cortex dans les Meningites Aigues. By PIERRE THOMAS. (Paris, J. B. Bailliere et Fils, 19, Rue Hautefeuille.)

IN this work the author maintains that in acute meningitis of specific infective origin, all the layers of the cortex are affected. The first elements to show any lesion are the larger and more highly specialised cortical cells. These show alterations before any change can be detected in the vessels, and with an intensity which appears to be in relation to the infectivity of the cause, the duration of the infection and, apparently, the size of the elements themselves. The stages of such changes are, first, alteration in position, then of the shape, of the nucleus, pericellular infiltration, intra-cellular infiltration, and, lastly, destruction of the element. Judging from the cases observed by the author, neuroglial changes are secondary and appear slowly. The symptoms due to these lesions, be they exaggeration or diminution of neuron function, are delirium, motor symptoms, disorders of speech, coma, and alteration of sensation. In the opinion of the author, the lesions which he describes begin in the gray matter, and extend to the vessels and meninges. He believes that the *rôle* of the vessels has been considerably exaggerated, if not misunderstood. In other words, he regards the presence of specific diseases, such as pneumonia and tubercle, as having a selective effect on the large and other pyramidal cells of the cortex.

DOUGLAS STANLEY.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

At the Clinical Meeting held at 11, Chandos Street, on Thursday, March 30, at 8.30 p.m., the following cases were shown :—

Dr. Ormerod.—(*a*) *Case resembling Friedreich's disease, but exhibiting extensive anæsthesia.*

[1] Male, aged 21, formerly a clerk.

Family history.—Eldest of four children, of whom the next in order, viz., a sister, aged 19, is said to be similarly affected.

History of illness.—Weak on his legs from an early stage. Upper limbs affected (difficulty in writing first noticed) for the last five years. Speech affected, and affection of limbs worse since an illness (said to be "phthisis") three years ago. Numbness and formication in limbs, two years. Loss of sensation of passing urine one year.

Condition on admission to the National Hospital (January 20, 1905).—Unsteady gait. Hardly able to walk without touching neighbouring objects. Romberg's symptom present. Unable to write more than a few words at a time (painful cramp sets in). Speech somewhat slurring and nasal. Knee-jerks absent. Pupils normal. Anæsthesia to touch and prick as shown in charts. Loss of taste in anterior two-thirds of tongue. No loss of thermal sense, but a bath at 100° produces sensation of scalding and formication.

(*b*) *Case for diagnosis, (possibly) progressive disease of pons and peduncles.*

[2] Man, aged 28, formerly a country policeman, previous health good, no history of syphilis.

In May, 1903, sudden attack, in which eyes were drawn to left, and face drawn to left (lesion in neighbourhood of right sixth nucleus ?), numbness left hand, giddiness and reeling to left.

Recovery in ten weeks; during recovery, diplopia, right eye turning inwards.

During the next ten months four further attacks followed, of diplopia associated with right facial palsy, all getting well, though his walking appears to have remained unsteady. The sight in the right eye has been bad since the first attack; for this there is a local cause, a large veil opacity in the vitreous.

February, 1904.—Attack in which left leg became weak, and left arm weak and shaky (but no return of diplopia or facial palsy). After this attack the change in his voice developed.

On admission to the National Hospital (August 5, 1904).—Ocular movements fair (? some weakness right external rectus). Some weakness right lower face (which passed off). Speech rather husky and squeaky. Weakness, tremors, inco-ordination in movements of left limbs. Tendency to fall to left. Some rigidity left leg; knee-jerk increased left side. Left ankle clonus, and plantar reflex extensor. Numbness left limbs, but no objective affection of sensation.

August 29.—Paralysis in district of *left third nerve trunk*, which passed off gradually.

September 23.—Vomiting and giddiness; cannot stand alone; falls to *right*. Position of head = occiput drawn down towards right shoulder and chin rotated to left. This passed off.

November. — Rhythmical oscillations of palate and vocal cords (and diaphragm ?) first observed.

December 26.—Vomiting and gastro-intestinal disturbances ascribed to Christmas dinner, but followed by :—

December 28.—A *second* attack of paralysis in the district of the *left third* nerve trunk.

Main features at present time (March 17, 1905) are :—Left hemiplegia incomplete. Irregular movements of left arm, which are, however, less marked than formerly. Incomplete ocular palsies = deficient movement of convergence; deficient upward movement, left. Peculiar explosive and squeaky speech; this has become more marked since admission. "Nystagmus" of palate and vocal cords.

Dr. GORDON HOLMES.—*Case of midbrain lesion with unilateral tremor.*

[3] A. V., aged 10 years, was admitted to the National Hospital in February, 1905.

Family history unimportant, previous health good.

Four years ago he became unsteady on his feet, and began to stagger like a drunken man. Gait has since steadily become worse. Has walked over to the left. Fifteen months ago his left arm was noticed to be weak and awkward, and a few months later the right arm began to shake.

Four years ago he also developed squint; the left eye turned outwards, and since then he has not been able to look up properly. During his illness frequent headache and vomiting, and his mental condition has deteriorated.

Sight and hearing normal, no ophthalmoscopic changes. Paralysis of upward movement of the eyes, weakness of their downward movements and of left internal rectus. Pupils almost inactive to light, but contract on accommodation. Head and eyes generally deviated to left. Tremor of right half of tongue. Irregular tremor of head, which is generally rotatory.

No wasting of muscles; slight rigidity of right limbs, but power of both right and left fair. Except when they are fully supported, and during sleep, there is constant tremor of the right limbs involving both distal and proximal muscles. It is fairly regular in rate and range, but intermittent and often rhythmical. The oscillations are somewhat coarse, and about four to five per second. Increased by excitement and on effort, and cannot be inhibited by patient. Intention tremor on volitional movement. There is a tendency to athetoid movements of left hand, but no ataxia. His gait is very ungainly, reeling and staggering.

There is no sensory change. The tendon reflexes are brisk, and the plantars of the extensor type.

There is considerable mental defect.

Dr. WILFRID HARRIS.—*A case of chronic chorea.*

[4] An engine driver, aged 63, while at work ten years ago strained his back, causing such pain in the left side of his back that he had to walk home doubled up. He has never walked properly since, and soon afterwards became unsteady in his gait. At the same time, though a teetotaller, his articulation became altered and slow, and he was often accused of being drunk. He has not been able to do any work for the last nine years.

There has been precipitancy of micturition, with occasional incontinence, for the last four years. His memory for recent events is not very good, and he has occasional difficulty in remembering where he is on waking. He knows of no other member of the family similarly affected, and he denies venereal disease.

There are now constant choreic movements of the face, head and limbs. The articulation is difficult to understand on account of its explosive character. The gait is markedly ataxic, and somewhat spastic. The grip is poor. The ataxy of the arms resembles that of Friedreich's disease. The left leg and the left arm are the weaker, and the head is usually slightly drawn down towards the left. The knee-jerks are brisk, L. > R., with slight left ankle clonus. Plantar responses are flexor. The eye movements and pupils are normal; no nystagmus. Visual fields and acuity normal. Discs normal.

The case appears to be a combination of spastic paralysis and chronic chorea, and might be labelled "spastic chorea." The history of onset and absence of heredity seem to exclude Huntingdon's chorea.

DR. A. F. VOELCKER.—*A case of acute ataxy (encephalitis cerebelli).*

[5] D. F., girl, aged 4½ years, was admitted to the Children's Hospital "for weakness and paralysis" on March 7, 1905.

On December 24, 1904, a rash like that of scarlet fever was noticed all over the body. The child became feverish and listless, and on the fifth day was in great pain, both legs were drawn up and the child was a little rigid. She then became unconscious for fourteen days, during which time there were occasional very slight twitchings of the face, and she occasionally put her hand up to the right side of the head. After the third week of illness she recovered consciousness, but has been unable to stand, feed herself, or speak properly since.

There has been neither otorrhœa nor vomiting.

The mother had chorea as a child, but beyond this the family history is unimportant.

State on admission.—Well grown, rather heavy aspect, lies down in bed and cannot sit up alone. She understands what is said to her. Speech very slow, no stammering or elision. Says very few things beyond her name, address, "yes" and "no." When sat up there is tremor of the head, which tends to fall forward. Cannot stand alone, but with support tends to exhibit some cross-legged progression. The feet are extended. No rigidity of limbs. Marked ataxy of both upper and lower limbs. Irregular choreiform movements of the hands. Marked intention tremor.

Cranial nerves.—No optic neuritis. No nystagmus. No squint or ocular palsy. Pupils equal and react to light and accommodation. No facial palsy. Hears well. Tongue protruded a little to the right.

Reflexes.—Plantar variable. Knee-jerks brisk. No ankle-clonus.

Sphincters.—No loss of control.

No evidence of disease in thorax or abdomen. Temperature 97 to 100°.

Progress.—Slight improvement in speech since admission.

Dr. T. D. Savill.—*Two cases of generalised tonic muscular spasm in middle-aged men, probably paralysis agitans.*

[6] (*a*) O. J. C., a schoolmaster, aged 38. The present illness began when he was 36, in the summer of 1902, with stiffness, slight shaking, and sometimes pain (chiefly in the arms), which was followed in the winter, 1902-1903, by stiffness and dragging of the legs. But he was able to play tennis and bicycle up to the summer of 1903, and he kept at work until Christmas, 1903.

When he first came under notice in June, 1904, there was a generalised stiffness of all the muscles, including those of the face and chest. Every movement was slow and deliberate, both feet were scraped along the ground, especially the left. The gaze and attitude were fixed, and he bent forward during progression. Expression mask-like. Small tremor of the hands, even when supported. Grip of hands about $\frac{5}{6}$ths of normal; muscular power good, but could not walk more than half a mile without increased stiffness and fatigue. Pupils normal, ocular movements stiff; no affection of cranial nerves. No sensory troubles. Skin of face glossy and chloasma across forehead. No history of syphilis.

The symptoms at the present time (March, 1905) remain much the same, but his power of moving his limbs has improved considerably under (1) galvanism to the muscles of the limbs for two months, and (2) high-frequency electricity—auto-condensation couch, effleuve and sparking to muscles. He can now walk two miles without undue fatigue.

[7] (*b*) H. F., a bricklayer, aged 44, came to the West End Hospital March 7, 1905, complaining of *stiffness* and loss of power in the arms and legs, which had started gradually eighteen months before. He said that the hands were first affected so that he could not hold the bricks, and soon afterwards the legs became involved. Nine months ago he first noticed a difficulty in speaking when he was excited.

Now there is a generalised muscular stiffness and slowness of movement. There is an uncertainty in the movements rather than an actual tremor. The gaze and attitude are fixed. The gait is stiff, and he has unsteadiness in turning, but there is no Rombergism or definite inco-ordination. Patellar reflexes increased, no Babinski's sign. The grasp is somewhat deficient, but muscular power everywhere is fair. The speech is deliberate and slightly explosive. The pupils react normally, and there is no ptosis, the fixity of the face suggests a rigidity of facial muscles. No obvious affection of the cranial nerves. There is no definite history of syphilis, but four of his sisters and one brother have died of consumption. The muscular power soon becomes exhausted, and the question of myasthenia gravis was considered, but there is no faradic reaction of exhaustion.

Dr. LEONARD GUTHRIE.—*Primary atrophy of optic nerves, with partial ophthalmoplegia and cerebellar gait.*

[8] W. L., aged 52, cabman, has complained of difficulty in walking for five or six years. Two and a half years ago he was seized whilst driving his cab by giddiness and temporary diplopia. Two years ago, whilst in bed, his left leg "kicked out" involuntarily, and he felt sensation of "pins and needles" in the fingers of his left hand. He did not lose consciousness. He has since complained of increasing weakness of legs, especially left, and unsteadiness and difficulty in walking.

Present condition.—Healthy-looking, ruddy-faced man. Heart, vessels, lungs and urine are normal. Pupils equal, reactions normal. There is some limitation of upward movements of eyes, and deficient convergence, with slow nystagmoid twitchings on external deviation. The optic discs are very pale, slightly excavated, the cribriform plates exposed, and vessels diminished in size. There is a ring of choroidal atrophy round the right disc. Vision is fairly good. The auditory and the other cranial nerves are unaffected. He has never suffered from headache or vomiting. Mind and intellect are clear. Tremors, ataxy, and pains are absent, sensation is normal everywhere. There is some weakness of the left hand-grasp. The knee-jerks are exaggerated, ankle-clonus is absent, plantar response is negative. Romberg's sign present. Gait is extremely unsteady. He reels and lurches from side to side with special tendency to veer towards the left. He drags his left foot, and dares not walk without support lest he should fall. The lesion is probably of the corpora quadrigemina.

Dr. HARRY CAMPBELL.

[9] (*a*) W. J. L., aged 73, bricklayer. Eighteen years ago tremor in thumb of left hand. Gradually increased, and about ten years ago tremor began in right hand, and interfered with writing. In all other respects seems to be healthy, and can walk long distances without fatigue. Knee-jerks active.

This patient is shown on account of the fact that his daughter (who is also shown to-night) has disseminated sclerosis.

[10] (*b*) J. A., aged 39. Four years ago began to see double. Three and a half years ago walking became difficult, and shortly after, the right hand began to tremble. Six months ago diplopia disappeared.

Present condition.—Pupils unequal. No nystagmus. Well-marked intention tremors especially right side. Knee-jerks greatly increased, ankle-clonus and Babinski's sign present. Speech, which was once very fluent, somewhat syllabic.

This patient is shown because her father (who is shown to-night) has had intention tremor for eighteen years.

DR. HEAD.—*A case of Huntingdon's Chorea.*

[11] H. S., aged 38. A little more than twelve months ago he noticed that his legs began to tremble. About seven months ago his speech became affected. He is married and has four healthy children. His brothers and sisters and their children are healthy. No similar condition has appeared in any of his relatives except in his mother's sister who is now 83 years of age. At the age of 40 she is said to have begun to be restless, as he is now, and to have remained so until the present time.

He is a well-developed man, with no disease in the heart or lungs; the urine is unaffected. He speaks like a man who has difficulty in controlling his tongue; each word is begun with some difficulty, and some words are slurred, especially when he is in the presence of strangers. The tongue is extremely unsteady. Face, hands, and legs are in constant restless movement, which is said to cease during sleep. Chest wall and diaphragm participate in these irregular movements. Tremor is absent. The knee-jerks are normal, and the toes go downwards when the sole of the foot is stimulated. The pupils act normally, and micturition is not affected.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Society can obtain the Index of "Brain" for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Society the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

Rules

AND

LIST OF OFFICE BEARERS
AND MEMBERS

OF THE

Neurological Society of the United Kingdom

1905

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1904

London
JOHN BALE, SONS AND DANIELSSON, Ltd.
83-91, GREAT TITCHFIELD STREET, OXFORD STREET, W.

1905

Office Bearers for the Year 1905.

President.

SIR JOHN BATTY TUKE, M.D., LL.D., F.R.C.P.Ed.

Vice-Presidents.

SIR THOMAS BARLOW, Bart., K.C.V.O., M.D., F.R.C.P.
CHARLES E. BEEVOR, M.D., F.R.C.P.

Council.

FLETCHER BEACH, M.B., F.R.C.P.
BYROM BRAMWELL, M.D., F.R.C.P.Ed.
JUDSON S. BURY, M.D., F.R.C.P.
J. MICHELL CLARKE, M.D., F.R.C.P.
JAMES S. COLLIER, M.D., F.R.C.P.
W. S. COLMAN, M.D., F.R.C.P.
PERCY KIDD, M.D., F.R.C.P.
H. W. PAGE, M.C., F.R.C.S.
G. NEWTON PITT, M.D., F.R.C.P.
S. J. SHARKEY, M.D., F.R.C.P.
R. PERCY SMITH, M.D., F.R.C.P.
ERNEST H. STARLING, M.D., F.R.S.

Treasurer.

JAMES TAYLOR, M.D., F.R.C.P.

Editor.

[illegible]RY HEAD, M.D., F.R.C.P., F.R.S.

Secretaries.

[illegible] M.D., F.R.C.P.
[illegible]UTHRIE, M.D., F.R.C.P.

Neurological Society of the United Kingdom.

REPORT OF THE COUNCIL FOR 1904.

FEBRUARY 16, 1905.

The council has much pleasure in reporting the continued prosperity of the Society, which now numbers 245 members.

The Presidential Address given on February 4 by Dr. Sharkey was fully reported in the Spring Number of "Brain." The abstract of the Proceedings shows the scope and character of the work of the Society during the past year. Three meetings were devoted to the illustration of single subjects in Neurology. All these evenings were unusually well attended.

Dr. Beevor resigns the Treasurership after 10 years' whole-hearted service, and the Council wishes to express to him its thanks for his devotion to the interests of the Society.

The Council also regrets that Dr. Percy Smith can no longer undertake the Editorship of "Brain." He assumed the post at a time when the Journal was heavily in debt. The long expected Index to "Brain" was far from completed, and much thankless toil was thrown upon him before he was able to issue it to the Society in 1902. Within four years, by unceasing care, he has so improved the finances of the Journal that there is now a substantial balance, and money can again be devoted to illustrations.

The Council wishes to express its thanks to the senior Secretary, Dr. Head, who retires after four years of most valuable service rendered to the Society. The Council has nominated Dr. Head as Editor of "Brain."

It is hoped that the prosperity of the Society will continue to increase, and so enable it to maintain the present high reputation of its Journal, "Brain."

Proceedings of the Society during the Year 1904.

February 14.—Dr. S. T. SHARKEY, Inaugural Address, "Hysteria and Neurasthenia."

March 17.—Dr. ALFRED W. CAMPBELL, "Histological studies in cerebral localisation."

April 21.—Clinical Meeting. Subject, "Tremor." Dr. JAMES TAYLOR, "Cases of Tremor." Dr. LEONARD GUTHRIE and Dr. RAYNER BATTEN, "Intention tremors, most marked on right side, associated with paresis of left superior rectus muscle, dilatation and inactivity to light of left pupil, unsteadiness of gait, marked 'Romberg's sign,' and active knee-jerks." Dr. HEAD and Mr. ERIC GARDNER, "Tremor in a case of family spastic paralysis." Dr. HEAD, "Tremor from a lesion, possibly of optic thalamus." Dr. LEONARD WILLIAMS (1), "Case of tremor in boy"; (2), "Generalised tremor associated with tabes"; (3), "Tremor of unusual distribution associated with Parkinson's disease." Dr. GEORGE JOHNSTON, "A case of paralysis agitans with intention tremor." Dr. E. FARQUHAR BUZZARD, "Case of tremor in boy." Dr. WILFRED HARRIS, "Case of familial tremor." Dr. T. GRAINGER STEWART, "Case of tremor."

July 14.—Clinical Meeting. Subject, "Muscular atrophy." (1) *Muscular Atrophy of Spinal Cord Origin.*—Dr. HENRY HEAD, "Widespread muscular wasting of both upper extremities with increased knee-jerks and flexor toe response." Dr. FERRIER (1), "Amyotrophic lateral sclerosis (upper arm type);" (2) "Progressive muscular atrophy (? early tabes dorsalis.") (2), *Peroneal Type.* Dr. HENRY HEAD (1), "A case of peroneal muscular atrophy"; (2), "A case of advanced peroneal atrophy." (3), *Neuritic Type.*—Dr. HENRY HEAD (1), "Muscular wasting from brachial neuritis"; (2), "A case of atrophy of the lower portion of the trapezius." (4), *Anomalous Cases.*—Dr. ORMEROD, "? Early syringomyelia or neuritis." (5), *Syringomyelia.*—Dr. FARQUHAR BUZZARD, "Case of syringomyelia. Dr. WILFRED HARRIS, "Case of syringomyelia."

October 27.—Clinical Meeting. Dr. FERRIER and Mr. BALLANCE, "Case of recovery after operation for cauda equina lesion." Dr. FERRIER and Sir V. HORSLEY, "Case of recovery after operation for tumour of cauda equina." Dr. COLMAN, "Case of myasthenia gravis with prominent bulbar symptoms." Dr. ALBERT WILSON, "Progressive muscular atrophy with arthritic changes." Mr. CHARLES BALLANCE (1), "A case of facial palsy treated by hypoglosso-facial and gustatorio-hypoglossal anastomosis"; (2), "A case of facio-accessory anastomosis for facial palsy, with recovery of dissociated movement." Dr. ALDREN TURNER, "Case of facio-accessory anastomosis." Mr. HAMILTON BALLANCE, "A case of facio-accessory anastomosis for facial palsy." Dr. PURVES STEWART and Mr. LENTHAL CHEATLE, "Case of facio-hypoglossal anastomosis." Mr. CHARLES BALLANCE, "A case of temporo-sphenoidal abscess with loss of the sense of smell." Dr. BEEVOR, "Pseudo-bulbar paralysis from bilateral hemiplegia." Dr. RISIEN RUSSELL, "Case of pseudo-bulbar paralysis (double hemiplegia)."

December 1st.—Pathological Meeting. Dr. DAVID ORR, a discussion on "Lesions of the posterior column of the spinal cord in diseases other than tabes dorsalis."

BALANCE SHEET, 1904.

NEUROLOGICAL SOCIETY.

Dr. — **Cr.**

RECEIPTS.	£	s.	d.
To Balance	62	0	10
Subscriptions	248	14	6
Half-Share profit on "Brain"	50	10	6
Contribution of Messrs. Macmillan towards cost of Illustrations for 1904	16	16	10
	£378	2	8

EXPENDITURE.	£	s.	d.	£	s.	d.
By Messrs. Macmillan for publishing "Brain," 1904 ...				148	8	3
Stationery and Printing—						
Bale and Danielsson	£33	17	3			
Pulman		11	0			
				34	8	3
Illustrations—Contributors of Papers	£49	0	0			
" Bale and Danielsson ...	38	8	3			
				87	8	3
Microscopes—Baker and Son				3	5	0
Lantern—Hawes				1	10	0
Hire of Rooms and Refreshments— Medical Society -				21	15	0
Petty Cash				2	13	4
Balance				78	14	7
				£378	2	8

Account for Publishing Index to "Brain," Vols. I.—XXIII.

	£	s.	d.
To Sale of Index (less Commission)	3	15	6
Balance against	50	14	6
	£54	10	0

	£	s.	d.
By Messrs. Macmillan on Index Account	54	10	0
	£54	10	0

General Statement.

	£	s.	d.
To Balance on General Account	78	14	7
	£78	14	7

	£	s.	d.
By Messrs. Macmillan on Index Account	50	14	6
Balance	28	0	1
	£78	14	7

Audited and found correct, January 9, 1905
H. CAMPBELL THOMSON.
J. S. COLLIER.

CHARLES E. BEEVOR, *Treasurer.*

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Guinea, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, the Editor of "Brain," and twelve Councillors, of whom not less than three shall be extra-Metropolitan Members.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years, one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six Ordinary Meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by Henry Head, M.D., F.R.S.

HONORARY MEMBERS.

Professor Wundt, Leipzig.
„ Hitzig, Halle.
Sir Samuel Wilks, Bart., London.
Dr. J. Hughlings Jackson, London.

CORRESPONDING MEMBERS.

Professor Dejerine, Paris.
„ Erb, Heidelberg.
„ Golgi, Pavia.
„ Grasset, Montpellier.
„ Luciani, Rome.
„ Munk, Berlin.
„ Obersteiner, Vienna.
„ A. Pick, Prague.
„ Retzius, Gustaf, Stockholm.
„ Ribot, Paris.
Dr. Weir Mitchell, Philadelphia.
Dr. Allen Starr, New York.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886	J. Hughlings Jackson, M.D., F.R.S.
1887	Sir Samuel Wilks, Bart., M.D., F.R.S.
1888	Sir James Crichton-Browne, M.D., F.R.S.
1889	Jonathan Hutchinson, F.R.S.
1890	Thomas Buzzard, M.D.
1891	John S. Bristowe, M.D., F.R.S.
1892	H. Charlton Bastian, M.D., F.R.S.
1893	Edward A. Schäfer, F.R.S.
1894	David Ferrier, M.D., F.R.S.
1895	Sir William H. Broadbent, Bart., M.D., F.R.S.
1896	Alexander Hill, M.A., M.D.
1897	George H. Savage, M.D.
1898	Sir Victor Horsley, M.B., F.R.C.S., F.R.S.
1899	W. H. Gaskell, M.D., F.R.S.
1900	Augustus Waller, M.D., F.R.S.
1901	W. Julius Mickle, M.D.
1902	Herbert William Page, M.A., F.R.C.S.
1903	J. N. Langley, D.Sc., F.R.S.
1904	S. J. Sharkey, M.D.

LIST OF HUGHLINGS JACKSON LECTURERS.

1897	J. Hughlings Jackson, M.D., F.R.S.
1900	Geheimrath Professor Hitzig, Halle.
1903	Sir William H. Broadbent, Bart., M.D., F.R.S.

, Members are requested to communicate with the Secretaries when corrections are necessary.

—:o:—

EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.
Pres., President.
V.-P., Vice-President.
Tr., Treasurer.
Sec., Secretary.
C., Member of Council.
H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected

1899 Abrahams, Bertram, M.B., M.R.C.P., 14, Welbeck Street, W.
1897 Anderson, H. Kerr, M.D., B.Sc., Whitefield, Great Shelford, Cambs.
1894 Andriezen, W. Lloyd, M.D., 7, Apsley Terrace, Acton, W.
1903 Armour, D. J., M.B., F.R.C.S., 89, Harley Street, W.
1904 Ascherson, W. Lawrence, M.B., M.R.C.P., St. George's Hospital, W.

1891 Ballance, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897-1900.)
O.M. Barlow, Sir Thomas, Bart., K.C.V.O., M.D., F.R.C.P., 10, Wimpole Street, W. (V.-P., 1904-05.)
1902 Barnes, A. Stanley, M.D., 141, Great Charles Street, Birmingham.
1897 Barratt, J. O. Wakelin, M.D., F.R.C.S., Bertheaustrasse 6, Göttingen.
O.M. Bastian, H. Charlton, M.D., F.R.C.P., F.R.S., 8a, Manchester Square, W. (Pres., 1892; V.-P., 1891; C., 1886-89, 1893-95.)
1897 Batten, Frederick E., M.D., F.R.C.P., 33, Harley Street, W. (Sec., 1903-05.)
1903 Bayliss, W. M., M.A., D.Sc., F.R.S., St. Cuthbert's, West Heath Road, Hampstead.
O.M. Beach, Fletcher, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey. (C., 1903-05.)
1903 Beddard, A. P., M.D., F.R.C.P., 44, Seymour Street, Portman Square.
O.M. Beevor, C. E., M.D., F.R.C.P., 135, Harley Street, W. (Tr., 1894-1905; V.P., 1905; C., 1893.)
1903 Berry, George A., M.B., F.R.C.S.Edin., 31, Drumsheugh Gardens, Edinburgh.
1898 Bolton, Joseph Shaw, M.D., B.S., B.Sc., East Sussex County Asylum, Hellingly, S.O., Sussex.
1896 Bond, C. Hubert, M.D., D.Sc., London County Colony for Epileptics, Ewell, Surrey.
1899 Bond, J. W., M.D., B.S., 26, Harley Street, W.
1892 Bowlby, Anthony A., C.M.G., F.R.C.S., 24, Manchester Square, W.
1898 Boyce, Rubert W., M.B., F.R.S., University College, Liverpool.
1889 Bradford, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 8, Manchester Square, W.

Elected	
O.M.	Bramwell, Byrom, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894, 1903-05.)
1899	Bramwell, Edwin, M.B., C.M., M.R.C.P., 23, Drumsheugh Gardens, Edinburgh.
1895	Bramwell, John Milne, M.B., C.M., 33, Wimpole Street, W.
1892	Bristowe, Hubert Carpenter, M.D., Wrington, Somerset.
O.M.	Broadbent, Sir Wm. H., Bart., K.C.V.O., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.-P., 1893; C., 1886-87, 1896.)
1894	Brodie, T. Grigor, M.D., F.R.S., 4, Lancaster Terrace, N.W.
1897	Brown, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.
1888	Brown, Sanger, M.D., 100, State Street, Chicago.
O.M.	Bruce, Alex., M.D., F.R.C.P.Ed., 8, Ainslie Place, Edinburgh. (C., 1895.)
O.M.	Bruce, J. Mitchell, M.D., F.R.C.P., 23, Harley Street, W.
O.M.	Brunton, Sir T. Lauder, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896-98.)
1895	Bryan, Frederick, M.B., Horton House, Horton, Epsom.
1895	Bryant, John Henry, M.D., F.R.C.P., 8, Mansfield Street, Portland Place, W.
1903	Bubb, William, M.R.C.S., L.R.C.P., County Asylum, Powick, Worcester.
1890	Bury, Judson Sykes, M.D., F.R.C.P., 10, St. John Street, Manchester. (C. 1905.)
1900	Buzzard, E. Farquhar, M.D., M.R.C.P., 33, Harley Street, W.
O.M.	Buzzard, Thomas, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.-P., 1888-89; C., 1886-87, 1891-95.)
1898	Caley, H. A., M.D., F.R.C.P., 24, Upper Berkeley Street, W.
1903	Campbell, A. W., M.D., County Asylum, Rainhill, Lancs.
1898	Campbell, Harry, M.D., F.R.C.P., 23, Wimpole Street, W.
1899	Chambers, James, M.A., M.D., The Priory, Roehampton.
1891	Clarke, J. Michell, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol. (C., 1903-05.)
1903	Clouston, T. S., M.D., F.R.C.P.Ed., Royal Asylum, Morningside, Edinburgh.
1892	Cole, Robert Henry, M.D., M.R.C.P., 48, Upper Berkeley Street, Portman Square.
1900	Cole, Sydney John, M.O. Wilts County Asylum, Devizes.
1899	Collier, J. S., M.D., B.Sc., F.R.C.P., 57A, Wimpole Street, W. (C., 1905.)
1890	Colman, W. S., M.D., F.R.C.P., 9, Wimpole Street, W. (Sec., 1899-1902. C., 1903-05.)
1890	Corner, Harry, M.D., Brook House, Southgate, Middlesex.
1899	Coupland, Sidney, M.D., F.R.C.P., 16, Queen Anne Street, W.
1901	Cox, Joshua John, M.D., St. Ronan's, Clarendon Road, Eccles, Manchester.
1901	Craig, Maurice, M.D., M.R.C.P., Bethlem Royal Hospital, S.E.
1898	Darley-Hartley, W., M.D., M.R.C.P., Main Road, Sea Point, Cape Town, South Africa.
1895	Davidson, Andrew, M.D., Callan Park, Sydney, N.S.W.
1897	Dawson, William R., M.D., F.R.C.P.I., Farnham House, Finglas, co. Dublin, Ireland.

Elected

1898 Dodd, Henry Work, F.R.C.S., 136, Harley Street, W.

O.M. Dreschfeld, Julius, M.D., F.R.C.P., Stanley House, Wilmslow Road, Withington, Manchester. (V.-P., 1898-99.)

O.M. Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1903 Dudgeon, L. S., M.R.C.P., 6, Powis Gardens, London, W.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

1899 Fawcett, John, M.D., F.R.C.P., 66, Wimpole Street, W.

O.M. Ferrier, David, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1886; V.-P., 1892-3; C., 1886, 1890-95.)

1903 Flashman, J. F., M.B., Ch.M., B.Sc., Totnes, Temple Street, Petersham, Sydney, Australia.

1903 Fleming, Robert, M.D., F.R.C.P.Ed., 10, Chester Street, Edinburgh.

1903 Fletcher, H. Morley, M.D., F.R.C.P., 98, Harley Street, W.

O.M. Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.-P., 1897-98; C., 1896, 1900.)

1903 Gibson, G. A., M.D., D.Sc., F.R.C.P.Ed., 3, Drumsheugh Gardens, Edinburgh.

1904 Giglioli Guiolo T., M.D., M.R.C.P., Via Farinata Degli Ulorti, 4, Florence.

O.M. Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1902 Goldschmidt, Oscar Bernard, M.B., Bishopstone House, Bedford.

1899 Goodall, Edwin, M.D., F.R.C.P., Joint Counties Asylum, Carmarthen.

1897 Gossage, Alfred Milne, M.B., F.R.C.P., 54, Upper Berkeley Street, W.

1892 Gotch, Francis, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 Grant, J. Dundas, M.D., F.R.C.S., 18, Cavendish Square, W.

O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899-1900.)

1895 Guthrie, Leonard, M.D., F.R.C.P., 15, Upper Berkeley Street, W. (C., 1904; Sec. 1905.)

1902 Hall, W. Hamilton, M.R.C.S., Fordcombe, Tunbridge Wells.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., Church Cottage, 17, Marylebone Road, N.W.

O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 Hanbury, William R., M.R.C.S., L.R.C.P., West Ham Borough Asylum, Goodmayes, Ilford.

1904 Harris, David, F., M.D., The University, St. Andrews.

1897 Harris, Wilfred J., M.D., M.R.C.P., 37, Queen Anne Street, W.

1898 Haslett, John Hanfield, M.R.C.S., L.R.C.P., Halliford House, Sunbury-on-Thames.

1891 Hawkins, Herbert Pennell, M.D., F.R.C.P., 56, Portland Place, W.

O.M. Haycraft, J. B., M.D., D.Sc., F.R.S.E., University College of South Wales, Cardiff.

1891 Head, Henry, M.D., F.R.C.P., F.R.S., 143, Harley Street, W. (C., 1900; Sec., 1901-1904; Editor of "Brain," 1905.)

O.M. Hill, Alex., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.-P., 1895; C., 1892-94.)

1905 Hobhouse, Edmund, M.D., F.R.C.P., 12, Second Avenue, Brighton (Hove).

Elected

1903 Holmes, Gordon M., M.B., B.Ch., National Hospital, Queen Square, W.C.

O.M. Horsley, Sir Victor, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.-P., 1896-97; C., 1892-93, 1899.)

1903 Howland, Goldwin, W., M.B., M.R.C.P., c/o Dr. Sheraton, Wyckliff College, Toronto, Canada.

1889 Huggard, William R., M.D., F.R.C.P., Davoz Platz, Switzerland.

1896 Hulme, George Frederick, M.B., C.M., South View, Montague Road, Felixstowe.

1904 Humphreys, F. R., M.R.C.S., L.R.C.P., 27, Fellows Road, South Hampstead.

1889 Humphry, Laurence, M.D., F.R.C.P., Lensfield, Cambridge.

1903 Hunter, Walter K., M.D., D.Sc., 7, Woodside Place, Glasgow.

1901 Hunter, William, M.B., C.M., Government Bacteriological Laboratory, Hong Kong.

1903 Hunter, William, M.D., F.R.C.P., 103, Harley Street, W.

O.M. Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.-P., 1887-88; C., 1886, 1890.)

1889 Hyslop, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.

O.M. Jackson, J. Hughlings, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897: C., 1887, 1892.). Hon. Memb., 1904.

O.M. Jessop, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 Johnston, George, M.D., M.R.C.P., 3, Montagu Place, Montagu Square, W.

1903 Jones, A. E., M.B., B.Sc., University College Hospital, Gower Street.

1892 Jones, Henry Lewis, M.D., F.R.C.P., 143, Harley Street, W.

1894 Jones, Robert, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

1904 Jones, Robert, F.R.C.S.Ed., 11, Nelson Street, Liverpool.

1902 Kelynack, T. N., M.D., M.R.C.P., 120, Harley Street, W.

1894 Kidd, Percy, M.D., F.R.C.P., 60, Brook Street, W. (C., 1904.)

1890 Langdon, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 Langdon-Down, Reginald L., M.B., M.R.C.P., 47, Welbeck Street, W.

O.M. Langley, J. N., M.A., D.Sc., F.R.S., Hedgerley Lodge, Madingley Road, Cambridge. (Pres., 1903; V.-P., 1901-1902; C., 1898-99, 1904.)

1888 Lawford, J. B., M.D., F.R.C.S., 99, Harley Street, W. (C., 1902-3.)

O.M. Lees, D. B., M.D., F.R.C.P., 22, Weymouth Street, W. (C., 1901-1903.)

1897 Lewis, H. Wolseley, M.R.C.S., L.R.C.P., London County Asylum, Banstead, Surrey.

1904 Lindsay, J. A., M.D., F.R.C.P., 13, College Square E., Belfast.

1897 Loewenthal, Max, M.D., M.R.C.P., 126, Princes' Road, Liverpool.

1899 Lord, John R., M.B., C.M., London County Asylum, Bexley, Kent.

1898 McAldowie, Alexander M., M.D., 6, Brook Street, Stoke-on-Trent.

1898 MacCormac, John M'Gee, M.D., 73, Victoria Place, Belfast.

1891 Macdonald, Peter William, M.D., C.M., The County Asylum, Dorchester, Dorset.

Elected

1902 McDougall, Wm., M.B., Weald Mount, Haslemere.
1902 Mackenzie, James, M.D., Bank Parade, Burnley.
O.M. Mackenzie, Sir Stephen, M.D., F.R.C.P., 31, Cavendish Square, W. (C., 1896-98.)
1896 Mackintosh, Ashley W., M.A., M.D., 9, Bon Accord Square, Aberdeen.
1903 Macnamara, E. D., M.B., B.C., 45, Campden House Road, W.
O.M. Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)
O.M. Mann, J. Dixon, M.D., F.R.C.P., 16, St. John Street, Manchester.
1902 Mantle, Alfred, M.D., M.R.C.P., Savile Place, Halifax.
1901 Marris, W. A., M.D., Springfield Road, King's Heath, Birmingham.
1894 Martin, Sidney, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.
1891 May, W. Page, M.D., F.R.C.P., 9, Manchester Square, W.
1890 Menzies, William Francis, M.D., B.Sc., M.R.C.P., Cheddleton Asylum, Leek, Staffordshire.
O.M. Mercier, C., M.B., F.R.C.P., Flower House, Southend, Catford, S.E. (C., 1899-1902.)
1897 Meyer, Adolf, M.D., LL.D., Pathological Institute, New York State Hospital, Ward's Island, N.Y., U.S.A.
O.M. Mickle, W. Julius, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (Pres., 1901; V.-P., 1900; C., 1895-97, 1902-4.)
1905 Milne, Robertson Chas. John, I.M.S., M.B., C.M., c/o Inspector General of Civil Hospitals, Bengal, Calcutta.
1895 Moore, Norman, M.D., F.R.C.P., 94, Gloucester Place, W.
O.M. Morris, Malcolm, F.R.C.S.Ed., 8, Harley Street, W.
1902 Morrison, C. S., L.R.C.P.E., County and City Asylum, Hereford.
1888 Mott, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-98; C., 1899-1902.)
1901 Mould, George Wm., M.R.C.S., Cheadle Royal Asylum, Manchester.
1899 Mould, Gilbert E., M.R.C.S., L.R.C.P., The Grange, nr. Rotherham.
1894 Murray, George R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne.
1889 Murray, H. Montague, M.D., F.R.C.P., 25, Manchester Square, W.
1904 Myers, Charles S., M.D., Melrose, Grange Road, Cambridge.

1904 Neild, Newman, M.D., Richmond Hill, Clifton, Bristol.
O.M. Nettleship, Edward, F.R.C.S., Shottermill, Haslemere, Surrey.
1902 Newington, Hayes, F.R.C.P.Ed., The Gables, Ticehurst, Sussex.
O.M. Nicolson, David, C.B., M.D., M.R.C.P.Ed., Balgownie, Guildford, Surrey.
1888 Niermeyer, J. H. H., M.D., Amsterdam.
1904 Nolan, M. J., L.R.C.P., L.R.C.S.I., Down District Asylum, Downpatrick.
1888 Norman, Conolly, F.R.C.P.I., F.R.C.S.I., St. Dymphna's, N. Circular Road, Dublin.

1887 Ogilvie, George, M.B., M.R.C.P., 22, Welbeck Street, W.
1887 Oliver, Thomas, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.
O.M. Orange, Wm., C.B., M.D., F.R.C.P., Oakhurst, Godalming, Surrey.
O.M. Ormerod, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)

Elected	
1899	Orr, David, M.D., County Asylum, Prestwich, Manchester.
1904	Owen, Arthur Geoffry, M.D., B.Sc., c/o Prof. Marich, Chanoury University, Melbourne.
O.M.	Page, Herbert William, M.A., F.R.C.S., 146, Harley Street, W. (Pres., 1902; V.-P., 1900-1; C., 1891-92, 1903-05.)
1903	Palmer, Fredk. S., M.D., M.R.C.P., 19, Harley Street, W.
1895	Parkinson, John Porter, M.D., M.R.C.P., 57, Wimpole Street, W.
1901	Parsons, John Herbert, D.Sc., F.R.C.S., 27, Wimpole Street, W.
1901	Passmore, E. S., M.D., M.R.C.P., Croydon Asylum, Warlingham, Surrey.
1896	Paterson, Donald Rose, M.D., M.R.C.P., 15, St. Andrew's Crescent, Cardiff.
1904	Paton, Leslie, J., M.B., F.R.C.S., 1, Spanish Place, W.
1899	Piper, Francis P., M.B., M.R.C.S., L.R.C.P., King's Leigh, Tankerton, Whitstable.
1888	Pitt, G. N., M.D., F.R.C.P., 15, Portland Place, W. (C., 1903-05).
1899	Rambaut, Daniel F., M.D., B.Ch., County Asylum, Bicton Heath, Shrewsbury.
1894	Ransom, William B., M.D., F.R.C.P., The Pavement, Nottingham.
1890	Rayner, Henry, M.D., M.R.C.P.Ed., 16, Queen Anne Street, W.
O.M.	Reid, E. W., M.B., F.R.S., University College, Dundee.
1897	Reissmann, Charles H., M.B., B.Sc., St. Peter's, Adelaide, South Australia.
1893	Rennie, George E., M.D., M.R.C.P., College Street, Sydney.
1895	Reynolds, Ernest S., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.
1891	Rivers, W. H. R., M.A., M.D., F.R.C.P., St. John's College, Cambridge. (C., 1897-98.)
1898	Robertson, William Ford, M.D., 7, Hill Square, Edinburgh.
1892	Rowe, Edmund Lewis, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.
1901	Rows, R. G., M.D., County Lunatic Asylum, Lancaster.
1892	Ruffer, Marc Armand, M.D., Ramleh, Egypt.
1903	Russell, A. E., M.D., M.R.C.P., 9, Wimpole Street, W.
1889	Russell, J. S. Risien, M.D., F.R.C.P., 44, Wimpole Street, W. (Sec., 1896-1900; C., 1901-1904.)
1899	Russell, J. W., M.D., M.R.C.P., 72, Newhall Street, Birmingham.
1901	Salaman, Redcliffe N., M.A., M.B., Frognal End, Frognal Gardens, Hampstead.
O.M.	Sanderson, Sir J. Burdon, Bart., M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.
O.M.	Savage, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres., 1897; V.-P., 1895; C., 1886-90, 1898.)
1888	Savill, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.
O.M.	Schäfer, E. A., F.R.S., University of Edinburgh. (Pres., 1893; V.-P., 1892; C., 1886-98.)
1901	Schorstein, Gustave, M.D., F.R.C.P., 11, Portland Place, W.
O.M.	Semon, Sir Felix, C.V.O., M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899-1901.)
1898	Seward, Wm. Joseph, M.B., Colney Hatch Asylum, N.

Elected

O.M. Sharkey, S. J., M.D., F.R.C.P., 22, Harley Street, W. (Pres., 1904; V.-P., 1902-3; C., 1899-92, 1905.)

O.M. Sherrington, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)

1894 Shuttleworth, George E., M.D., Ancaster House, Richmond.

1900 Singer, H. D., M.D., M.R.C.P., Macoagne Buildings, Omaha, Neb., U.S.A.

1895 Smith, F. J., M.D., F.R.C.P., 138, Harley Street, W.

1887 Smith, R. Percy, M.D., F.R.C.P., 36, Queen Anne Street, W. (Editor of "Brain," 1901-1904; C. 1905.)

1895 Spicer, W. T. Holmes, M.B., F.R.C.S., 5, Wimpole Street, W.

1901 Stafford, William, M.B., 110, Mansfield Road, Nottingham.

1895 Stanley, Douglas, M.D., M.R.C.P., 21, Broad Street, Birmingham.

1895 Stansfield, Thos. E. K., M.B., London County Asylum, Bexley, Kent.

1892 Starling, Ernest Henry, M.D., F.R.C.P., F.R.S., 40, West End Lane, Hampstead (C., 1904-05.)

1901 Steell, Graham, M.D., F.R.C.P., 96, Mosley Street, Manchester.

1897 Stewart, Purves, M.A., M.D., M.R.C.P., 7, Harley Street, W.

1903 Stewart, T. Grainger, M.B., B.Ch., M.R.C.P., National Hospital, Queen Square, W.C.

1897 Stirling, Wm., M.D., D.Sc., Owens College, Manchester.

1897 Stoddart, Wm. H., M.D., M.R.C.P., Bethlem Royal Hospital, Lambeth, S.E.

1902 Stone, W. G., M.D., 93, Denmark Hill, S.E.

1902 Sullivan, Wm. Charles, M.D., H.M. Prison, Pentonville, N.

1904 Sutherland, Geo. Alexander, M.D., F.R.C.P., 73, Wimpole Street, W.

1888 Syers, Henry W., M.D., M.R.C.P., 75, Wimpole Street, W.

1898 Taylor, F. R. P., M.D., East Sussex Asylum, Hellingly, Sussex.

1891 Taylor, James, M.D., F.R.C.P., 49, Welbeck Street, W. (C., 1900-3 Tr., 1905.)

1898 Thomson, H. Campbell, M.D., F.R.C.P., 34, Queen Anne Street, W.

1889 Thorburn, Wm., F.R.C.S., Oxford Lodge, Victoria Park, Manchester. (C., 1901-1903.)

1892 Titchener, E. B., B.A., Ph.D., Cornell Heights, Ithaca, N.Y.

O.M. Tooth, H. H., C.M.G., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-99.)

1892 Trevelyan, E. F., M.D., B.Sc., F.R.C.P., 40, Park Square, Leeds.

1892 Tuckey, Charles Lloyd, M.D., 88, Park Street, Grosvenor Square, W.

1892 Tuke, Sir J. Batty, M.D., F.R.C.P.Ed., F.R.S.Ed., 20, Charlotte Square, Edinburgh. (V.P., 1903-04; Pres., 1905.)

1891 Tuke, Thomas Seymour, M.B., Chiswick House, Chiswick.

1891 Turner, William Aldren, M.D., F.R.C.P., 18, Harley Street.

1898 Turney, Horace G., M.D., F.R.C.P., 68, Portland Place, W.

O.M. Tweedy, John, F.R.C.S., 100, Harley Street, W.

1901 Vincent, Swale, M.B., Physiological Laboratory, University of Manitoba, Winnipeg, Canada.

1888 Voorthus, J. A., M.D., Apeldoorn, 542, Loulaan, Netherlands.

Elected	
O.M.	Waller, Augustus, M.D., F.R.S., 32, Grove End Road, N.W. (Pres., 1900; V.-P., 1899; C., 1894-97, 1901.)
O.M.	Warner, Francis, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.
1902	Warrington, W. B., M.D., M.R.C.P., 69, Rodney Street, Liverpool.
1904	Watson, Chalmers, M.B., F.R.C.P.E., 22, Coates Crescent, Edinburgh.
O.M.	Watteville, A. de, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896-1902; Sec., 1886-89; Editor of "Brain," 1880-1900.)
1897	Weber, F. Parkes, M.D., F.R.C.P., 19, Harley Street, W.
1897	West, Samuel, M.D., F.R.C.P., 15, Wimpole Street, W.
1897	White, Ernest W., M.B., M.R.C.P., City of London Asylum, Dartford.
O.M.	White, W. Hale, M.D., F.R.C.P., 65, Harley St., W. (C., 1897-1902.)
1894	Whiting, Arthur J., M.D., M.R.C.P., 142, Harley Street, W.
1889	Wiglesworth, Joseph, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.
1905	Wilkin, Griffith Charles, M.R.C.S., L.R.C.P.Lond., Conway House, Paignton, Devon.
1900	Williams, Leonard, M.D., M.R.C.P., 8, York Street, Portman Square, W.
1894	Williamson, Richard T., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.
1896	Wilson, Albert, M.D., 1, Belsize Park, N.W.
1903	Wilson, G. R., M.D., M.R.C.P.Ed., 8, Rutland Square, Edinburgh.
1905	Wilson, S. A. K., M.A., M.B., Ch.B., B.Sc., National Hospital, Queen Square, Bloomsbury.
1896	Wood, Guy M., M.B., 49, Gordon Square, W.C.
1889	Wood, T. Outterson, M.D., F.R.C.P.Ed., 40, Margaret Street, Cavendish Square, W.
1892	Woodhead, G. Sims, M.D., F.R.C.P.Ed., 6, Scroope Terrace, Cambridge.
1899	Wright, Hamilton, M.D., Pathological Institute, Selangor, Straits Settlement.
1898	Young, Robert Arthur, M.D., B.Sc., M.R.C.P., 41, Wimpole St., W.

[NOVEMBER, 1905.]

BRAIN.

PART II., 1905.

Original Articles and Clinical Cases.

THE AFFERENT NERVOUS SYSTEM FROM A NEW ASPECT.[1]

BY HENRY HEAD, M.D., F.R.S.,
Physician to the London Hospital.

The conclusions expressed are drawn from investigations carried out in conjunction with

W. H. R. RIVERS, M.D.,
Fellow of St. John's College, Cambridge,

AND

JAMES SHERREN, F.R.C.S.,
Assistant Surgeon to the London Hospital.

IT has long been recognised, by all who have interested themselves in the problems of sensation, that no view yet advanced of the structure and functions of the afferent nervous system is sufficient to explain obvious facts. The teaching of the anatomist throws little light on the difficulties with which the surgeon is confronted. On the other hand, it is difficult to reconcile the various views concerning the nature of common sensibility with the facts of clinical experience.

Such want of correspondence between observed facts and the prevailing general ideas showed that the distribution and function of the peripheral nerves required reconsideration. In the present paper we shall put forward a new view of the mechanism of sensation, based upon several different lines of research. If we may seem unduly to neglect the work of others, let it be remembered that this paper is introductory to a series of communications, each of which will deal with one aspect of the subject more exhaustively than is possible in a preliminary statement of a new hypothesis.

---

[1] The substance of this paper was delivered on May 23, 1905, before the Royal Medical and Chirurgical Society as the Marshall Hall address.

When the median nerve is divided, sensation is entirely lost over a considerable part of both the index and middle fingers. Over the palm, within the area said by the anatomists to be supplied by this nerve, sensation is usually diminished and not completely abolished. In a similar manner, division of the ulnar nerve produces complete insensibility of the little finger, and of a variable portion of the ulnar aspect of the palm; but partial loss of sensation is found over a larger area of the palm and the ulnar half of the ring finger. Such is the usual statement of surgeons and anatomists. When they are asked, why sensation is only partially lost over the palm, the usual answer is, "Because there the nerves overlap." But if each nerve occupies the territory of the other to an extent sufficient to prevent absolute loss of sensation over so large a portion of the palm, it is obvious that destruction of the ulnar nerve must cause some diminution of sensibility over the median half. This loss should vary exactly in proportion to the amount of sensation that remains, after the median has been destroyed. But the most careful examination of the hand fails to show the slightest diminution in sensation over the median half of the palm in consequence of division of the ulnar nerve. What has always been called the diminished sensibility produced by the division of a nerve is really a condition in which some kinds of sensibility are lost and others retained. Within such a region of altered sensibility all sensation to light touch is abolished. If, in a patient who has divided his ulnar nerve, the ulnar half of the palm of the hand is stimulated with cotton-wool, no sensation will be produced, while the lightest touch will be appreciated directly the line corresponding to the axis of the index finger is transgressed. If the area is large enough to apply a pair of compasses, it will be found that the patient is totally unable to appreciate two points two centimetres apart. Not only is sensation abolished to these tests, but careful examination shows that temperatures between 22° C. and 40° C. are not appreciated over this area. Thus, parts which have universally been considered to be areas of diminished sensibility turn out to be totally insensitive to certain higher forms of stimulation.

When the hand has settled down after the shock of the injury that has divided one or more of the nerves to the palm, it will be found that, although the area we have spoken of is totally insensitive to certain higher forms of stimulation, a stimulus producing pain, *e.g.*, a prick of a pin, causes a more unpleasant effect than over normal parts.

If the nerve has been united, sensation begins to return after a variable interval. The first sign of recovery is a gradual diminution in the extent of the area insensitive to pain and to all forms of heat and cold.

Finally, no part of the affected hand remains completely insensitive to all cutaneous stimuli. It is to the condition of a hand at this stage of recovery that we wish to draw particular attention. It might be supposed that, with the gradual disappearance of analgesia, an improvement would follow in the higher forms of sensibility. This is not so The boundary at which light touch is lost is as definite as in the days following the injury, although sensibility to pain, to heat and to cold, has vastly improved. In this condition the hand may remain for many months, before light touch begins to be appreciated over parts that lie within the borders of altered sensibility.

Closer examination of parts in this condition shows that, although the hand has become sensitive to pain and to temperature, this sensibility is strangely altered. A prick is appreciated, but produces a sensation that radiates widely over the affected area. It causes unnatural discomfort, and the patient has an uncontrollable desire to withdraw his hand. Moreover, although ice and water at a temperature of 50° C. are appreciated as cold and hot, intermediate degrees produce no sensation of temperature, and water at 25° C. or 26° C. may be indistinguishable from water at 40° C.

We assured ourselves of the truth of these conclusions during more than two years spent in watching patients who had come to the London Hospital on account of injuries to one or more peripheral nerves. But it became obvious, that in order that we might examine more exhaustively the sensory condition of parts that had been robbed of their nerve supply, it was necessary that the patient should be a trained observer, and the injury determined beforehand.

On April 25, 1903, the radial (ramus cutaneus n. radialis) and external cutaneous nerves were divided in the neighbourhood of my elbow, and after small portions had been excised, the ends were united with silk sutures. Before this operation, the sensory condition of the arm and back of the hand had been minutely examined, and the distance at which two points of the compass could be discriminated had been everywhere measured.

This operation produced loss of all forms of cutaneous sensibility over an extensive area on the radial half of the forearm and back of the hand. Stimulation with cotton-wool, the prick of a pin, the application of all forms of heat and cold, were unappreciated, and the two points of the compasses could not be discriminated, even when separated to the furthest extent possible. But if this part was touched with the point of a pencil, the head of a pin or even with the ball of the finger, the stimulus was at once appreciated, and the point of application localised with remarkable accuracy. (Plate I., fig. 1.)

We are thus face to face with the conclusion, that complete destruction of all the sensory nerves to the skin leaves the part sensitive to most of those stimuli commonly used by the physician and surgeon as a test of sensibility to touch. With the Graham-Brown æsthesiometer, an instrument which measures the appreciation of irregu-

PLATE I.

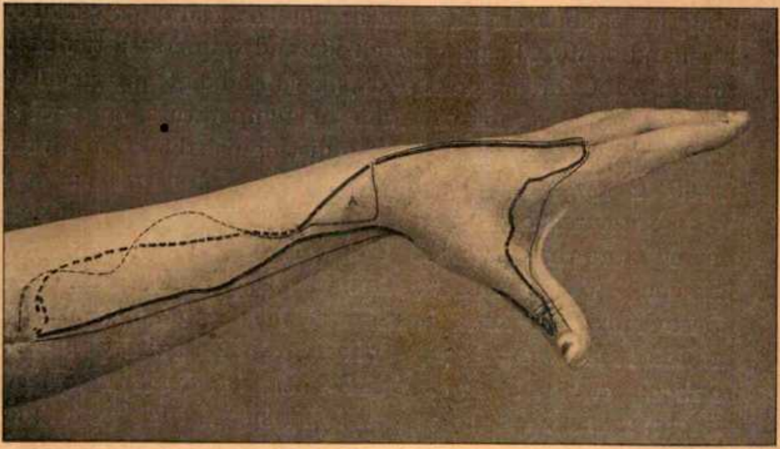


FIG. 1.

To show the loss of cutaneous sensibility produced by dividing the radial (ramus superficialis n. radialis) and external cutaneous nerves in the neighbourhood of the elbow. The thick line bounds the area insensitive to prick. The thinner line encloses the parts insensitive to cotton wool. Both the thick and the thin line are dotted wherever the borders of the area of loss of sensibility were not sharply defined.

The triangle marked **A** was insensitive to prick but sensitive to stimulation with cotton wool.

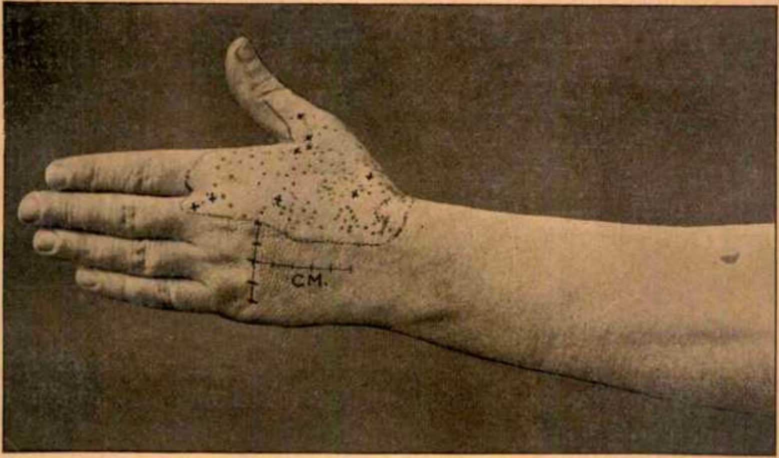


FIG. 2.

The area enclosed within the black line had become sensitive to prick, to water at 50° C. and to ice, but was insensitive to cotton wool, to water at 25° C. and to water at 38° C. The dots within this area are cold spots, the crosses mark the position of the hot spots. The two lines outside the affected area are divided into centimeters.

larities in an otherwise smooth surface, the hand that had been robbed of all its cutaneous sensibility was found to be actually more sensitive than a similar part on the normal side.

Since all the nerves had been divided which supplied the skin, the maintenance of this sensibility must have been due to afferent fibres running with motor nerves. Sherrington (1) has demonstrated the existence of such sensory fibres and traced them to the muscles, tendons and joints. By the operation on my arm, we had gained the unique opportunity of exposing a part, endowed only with deep sensibility, to a series of careful tests.

The peculiar aptitude, possessed by a part innervated solely by the afferent fibres of a muscular nerve, is the appreciation of all stimuli which produce deformation of structure. Pressure or any jarring of the skin was quickly appreciated in my case, and, on the whole, was localised with remarkable accuracy. But, when the hairs were pulled, the elevation of the skin produced no effect upon consciousness. Pressure, which had previously caused a sensation, was no longer appreciated when applied to the skin lifted from the subcutaneous structures to form a ridge. This showed that the sensibility to pressure was not due to nerves still remaining in the skin after the operation. Although pressure was localised with considerable accuracy, all sense of form and size was lost over the parts affected. The prick of a pin and the interrupted current were entirely unappreciated; but excess of pressure produced aching pain. When the pressure was produced by means of Cattell's algometer, it was found that pain was elicited with a smaller pressure of the instrument than on the sound side. The affected parts could be burnt without producing pain, and no sensation of cold was produced, even when the hand was frozen firmly by means of ethyl chloride.

This condition remained unaltered until seven weeks after the operation, when sensation of prick began to return on the arm. Six weeks later, there was no part of the forearm where prick could not be appreciated, and within 200 days from the time when the nerves were divided, even the back of the hand had become sensitive to this form of stimulation. Yet, for more than a year, both forearm and hand remained completely insensitive to light touch, and even now, more than two years after the operation, the hand has not completely regained its sensibility, when tested with cotton-wool and with the compasses.

Thus, we had ample opportunity of examining with care the sensory condition of a part sensitive to prick, but insensitive to light touch. We found that, when the forearm or hand was pricked, the pain produced was not localised, but radiated widely, and was not infrequently referred to some part at a distance from the point stimulated. Ice and water at 50° C. were appreciated, but minor degrees of temperature produced no effect upon consciousness. This peculiarity in the behaviour of the hand and forearm we found to be due to what are known as "cold" and "hot spots." (Plate I., fig. 2.)

Blix (2) first described the presence of "cold spots" in the skin, and his work was amplified by Goldscheider (3). To some observers, such as von Frey (4), all forms of sensation possessed by the skin are due to the existence of small areas of specific sensibility. So extreme a view has been accepted by few; some even doubt the very existence of temperature spots. By suitable methods spots can be demonstrated in the normal skin where cold alone can be appreciated; analogous spots, more sparsely scattered, can also be shown to be devoted entirely to sensations of heat. In the same way it would seem that there are spots peculiarly sensitive to the prick of a sharp needle; but the disturbance produced by their stimulation is so great, that they cannot be demonstrated with the same certainty as the spots devoted to sensations of temperature. Now *pari passu* with the return of sensibility to prick and to the extremes of heat and cold, these spots reappeared upon my arm and hand. But whereas, in the normal skin, the hot and cold spots are nothing more than minute areas peculiarly sensitive either to heat or to cold, set in a territory over which temperature stimuli can also be appreciated, the spots which made their reappearance on my arm during the first stage of recovery were set in an area insensitive to temperature stimulation. Thus, they were not only discoverable with unusual ease, but, since the only form of temperature sensation possessed by the recovering part was due to their presence, it was particularly easy to investigate their sensory peculiarities. The cold spots could be stimulated by any temperature below about 24° C.; but, whenever a spot reacted, what might be called an explosion of cold was produced, not localised at the point touched, but radiating widely, sometimes even to a very considerable distance. A small group of spots on the wrist always produced a sensation of cold in the forearm just below the fold of the elbow, and two spots in the forearm, when stimulated, evoked a sensation of cold in the thumb. The hot spots, more sparsely scattered, behaved in a similar manner. The lower limit of temperature to which they reacted varied from 38° C. to 45° C. One extremely sensitive spot even reacted to 37° C. But, whether these spots reacted slowly or briskly, the sensation was always one of widespread heat, and, until the painful limit of heat was reached, it mattered little at what temperature the stimulus was applied, provided it lay within the limits capable of stimulating these spots. The following experiment, which demonstrates this peculiarity, was many times repeated with the same results. A cold spot of unusual activity was stimulated by means of a copper cylinder of one millimetre diameter, cooled to the temperature of melting ice. This produced a sensation of cold. Water at 20° C. was placed in a test tube with a flat bottom of one centimetre diameter, and this was applied to the skin in such a way that it stimulated a constellation of spots, among which lay the spot originally stimulated. The sensation of cold produced by this stimulus was more intense than that produced by stimulating a single spot with a temperature considerably lower. Thus we come to the remarkable conclusion that the hot and cold spots are incapable of producing, in consciousness, graduated sensations of heat or of cold. Water at 20° C. can be made to appear colder than ice, provided the stimulus is so arranged that the former is applied over a considerably larger area than the latter. Such spots resemble in their action the cold alarms of our greenhouses. When the temperature falls below a certain amount, a bell is rung, but no indication is given of the extent to which the temperature has fallen.

Although we had peculiar difficulty in demonstrating the presence of similar spots for pain, the general behaviour of a part in this stage of recovering sensibility, closely resembles that of the hot and cold spots. Radiation takes place widely; a more intense stimulus is necessary to evoke pain, but when evoked, the pain is greater than over the normal skin. We also found that in this stage of recovery many of the hairs had gained a peculiar sensibility. When a hair on the normal skin is gently lifted, a sensation of touch is caused which is extremely well localised. But in the stage of recovery we are now discussing, the movement of the hairs produced a curious widespread formication, with the same reference to distant parts as in the case of temperature and pain.

However widespread the radiation may be to prick, to heat, to cold, or on touching the hairs, it is not fortuitous in its distribution. We found, by repeated experiment, that certain areas on the hand always caused radiation into some other part irrespective of the form of stimulation.

All these facts would seem to show that we are here face to face with an undiscovered form of sensibility, capable of producing qualitative changes in consciousness, but incapable of causing a quantitative change apart from the extent of area stimulated The position of the point stimulated cannot be recognised and each stimulus causes a widespread, radiating sensation, not infrequently referred to parts at a distance. To this form of sensibility we propose to give the name "*protopathic.*"

The return of protopathic sensibility brings a cessation of all those destructive changes in nutrition that occur in parts where the skin is insensitive. Ulcers form, as the consequence of burns or cuts, and do not heal so readily as on the normal skin. But such trophic changes are confined to parts insensitive to protopathic stimuli. With the return of protopathic sensibility, ulcers cease to form, and sores heal as readily as on the normal skin, although the parts remain insensitive to all the higher forms of stimulation, such as light touch. Thus a part supplied by protopathic sensibility alone grows, and is repaired, as easily as the normal skin.

After the affected part has remained for a variable period in this condition, it begins to become sensitive to light touch, and degrees of temperature, which produce the sensations called "warm" and "cool" on the normal skin, are again distinguished correctly from one another. With the gradual return of sensation, it again becomes possible to discriminate two points touching the skin at distances more nearly normal, and the widespread radiation, so characteristic of the first stage of recovery, ceases, and is replaced by an increasing accuracy of localisation. To this form of sensibility we propose to give the name "*epicritic*," since it is peculiarly associated with the localisation and discrimination of cutaneous stimuli.

So far we have demonstrated the existence in the skin of two forms of sensibility, but have brought forward no evidence to show that they depend upon anything more than modifications of the same system of nerve fibres and end organs.

I can now deal with a curious phenomenon that occurred in the case of my arm. Over the radial half of the dorsum of the wrist, a triangular area of skin became entirely insensitive to prick in consequence of the operation. But this same area remained sensitive to touches with cotton-wool, and also, in a limited degree, to warmth. The area was small, and its epicritic sensibility was of a low order, but in spite of these disadvantages, it became obvious, after repeated testing, that the area was capable of appreciating temperatures between 36° C. and 45° C. It was, however, entirely insensitive to temperatures of 50° C. and above. To ice and to all forms of cold, this part was equally insensitive. It would therefore seem that, by a fortunate chance in nerve distribution, we had divided those fibres which subserved protopathic sensibility, leaving untouched, at any rate, some of those which conducted the impulses of epicritic sensibility. Such an observation can only be explained by assuming that the two forms of sensibility depend upon two separate systems in the peripheral nerves. Experiments with so delicate a sensory change can only be carried out satisfactorily by frequent repetition, by selection of occasions when the patient is, from the sensory point of view, in excellent condition, and under the rigid check of a large number of controls. These conditions are rarely, if ever, satisfied during the examination of patients in whom the loss of sensation has been produced by accident. We have, however, seen this dissociation of sensibility in a patient where, during the removal of a small tumour from the nerves at the base of the brain, Sir Victor Horsley produced profound loss of sensation to prick over the one-half of the face, unaccompanied by an equivalent loss of sensibility to light touch. Here there could be no doubt that the patient could appreciate warmth, but not heat, and he many times stated that a temperature of 55° C. was neither hot nor cold, but that 43° C. was undoubtedly warm.

The mode of recovery of sensation after injury to a peripheral nerve also supports the view, that these two forms of sensibility depend upon separate structures. If the nerve has been completely divided, protopathic sensibility returns first, followed at a considerably later period by return of epicritic sensation. Provided the nerve has been completely divided, we have never seen the faintest sign of returning epicritic sensibility, unless sensation to prick had already shown material improvement. But, if the nerve is only bruised or injured, so that its continuity is functionally, but not structurally, destroyed, the two forms of sensibility may return *pari passu*.

Evidently, the two systems regenerate with unequal facility. The protopathic system regenerates more rapidly and with greater ease. It can triumph over want of apposition and the many disadvantages that are liable to follow traumatic division of a nerve.

Moreover, the length of the nerve to be regenerated makes relatively little difference to the time at which protopathic sensibility returns. Although the nerves in my arm were divided at a point at least 20 cm. above the wrist, recovery began in seven weeks and was completed, even over the hand, in twenty-nine weeks. This compares favourably with most of our instances of primary suture, in which the nerve was divided at the wrist. But this is in no way true of the epicritic system. Provided the wound is healthy, and the operation of the primary suture has been successfully performed, the length of time required for epicritic regeneration depends upon the distance of the wound from the periphery. And this is why, in my case, the period between the close of the first stage and the beginning of the second stage of recovery was unusually prolonged.

Every peripheral nerve contains in varying proportion the fibres subserving these two forms of sensibility. Let us consider for a moment their distribution in the nerves, the trunks and the roots which supply the upper limb.

To simplify what must of necessity be a somewhat complex statement, I will deal first with the supply of epicritic sensibility only. On the palm of the hand, the area supplied by the ulnar and median nerves overlaps to an extent less than one-half the breadth of the finger. Consequently, the borders of the insensitive area produced by division of one or other of these nerves is well defined. The back of the hand can be roughly divided into two halves, by a line running from the knuckle of the middle finger to the middle of the back of the wrist. On the ulnar side of this line, the hand is supplied by the ulnar and internal cutaneous, on the radial side by a combination of external cutaneous, the radial and the long cutaneous branch of the musculo-spiral. If we now include the forearm, it will be found that a line drawn up the flexor surface continuous with the axis of the ring finger, and up the extensor surface, continuous with the line just mentioned on the back of the hand, divides the whole of the forearm and hand into a pre-axial and a post-axial portion. Of these the post-axial portion is supplied by the ulnar and internal cutaneous, the pre-axial portion by the median, the radial, the external cutaneous, and the long branch of the musculo-spiral.

Another border, which has the same character as these two axial lines, is the boundary separating the distribution of the median from that of the group of nerves supplying the radial half of the dorsal surface of the hand.

Whenever division of any nerve branch causes loss of sensation to light touch along one of these lines, that border will be well defined. Division of one branch only will produce no definite area of anæsthesia, unless that area is bounded by one or more of these lines.

Thus, provided the peripheral nerves are gathered into certain groups, it may be said that from the point of view of light touch, and other forms of epicritic sensation, very little overlapping occurs. These groups are as follows: (1) The ulnar and internal cutaneous; (2) the median; (3) the remainder of the pre-axial group.

From this arrangement, the distribution of protopathic sensibility differs fundamentally. Enormous overlapping occurs, as we have already seen from a consideration of the analgesia caused by division of the median or of the ulnar nerves. Evidently, the peripheral nerves, looked at broadly, form the units of epicritic supply. On the contrary, from the protopathic point of view, no one nerve forms anything more than a tributary supply of an area innervated by a plexus of nerves, and, whenever a single peripheral nerve is destroyed in the upper limb, the loss of light touch always exceeds considerably the extent of the loss to prick. But, as soon as we have to deal with destruction of the cords of the brachial plexus, the extent of the analgesia almost equals that of the loss to light touch; and, when several posterior roots have been divided, the extent of the area insensitive to prick may actually exceed that insensitive to light touch. Thus it is evident that, whilst the unit of supply for epicritic sensibility, looked at broadly, lies in the peripheral nerves, the unit of protopathic supply lies in the posterior roots. The more nearly a peripheral nerve represents the supply of one or more posterior roots, the more definite will be the borders of the analgesia produced by dividing that nerve. The median nerve probably contains sensory fibres from the seventh and eighth cervical, and possibly even from the sixth cervical and first dorsal. Destruction of this nerve will therefore only cut off protopathic sensibility from the comparatively insignificant area to which all the fibres from these roots run in the one nerve. On the contrary, the distribution of the external popliteal, including its lateral cutaneous branch, corresponds closely to that of the fifth lumbar root. Consequently, destruction of this nerve produces a widespread loss of sensation to prick, with an extremely well-defined border on the shin and dorsal surface of the foot.

The sensory mechanism in the peripheral nerves is thus found to consist of three systems :—

(I.) Deep sensibility, capable of answering to pressure and to the movement of parts, and even capable of producing pain under the influence of excessive pressure, or when the joint is injured. The fibres, subserving this form of sensation, run mainly with the motor nerves, and are not destroyed by division of all the sensory nerves to the skin.

(II.) Protopathic sensibility, capable of responding to painful cutaneous stimuli, and to the extremes of heat and cold. This is the great reflex system, producing a rapid widely diffused response, unaccompanied by any definite appreciation of the locality of the spot stimulated.

(III.) Epicritic sensibility, by which we gain the power of cutaneous localisation, of the discrimination of two points, and of the finer grades of temperature, called cool and warm.

Let us now pass to the consideration of the arrangement of sensation in the central nervous system. The view I shall put forward is based upon the examination of a series of cases of hæmorrhage into the spinal cord, and of injuries affecting its substance, producing what is usually known as Brown-Séquard paralysis. With these we have compared the sensory changes in syringomyelia and tabes dorsalis. Now, all these conditions demonstrate that, as soon as a sensory impulse reaches its first junction in the spinal cord, it becomes shunted into tracts devoted to the conduction of impulses, grouped in a way different from that found in the peripheral nerves. It is no longer a question of protopathic, epicritic, or deep sensibility; the tracts in the central nervous system are devoted to the conduction of impulses concerned with pain, heat, cold, and touch.

Thus, in Brown-Séquard paralysis, motion is lost in the one limb, and all sense of pain, heat or cold, is abolished in the other. Careful examination of this loss of sensation shows that sensibility is equally lost to all forms of temperature stimulation, and that we have here to do with no such separation into extreme and intermediate degrees as exists in the peripheral nerves.

In the central nervous system, the impulses are co-ordinated and distributed, just as in the central office of a newspaper the various accounts of the same event, arriving by telephone, by tape, or by telegraph, are co-ordinated and distributed according to their subject-matter.

It has long been recognised, that the viscera are not endowed with the same sensibility as the skin, and some have even questioned whether they are sensitive at all. It is certain, from the observations of Lennander (5) and his school, that the patient shows no sign of pain when the gut is incised, or even when it is burnt; the liver is also apparently insensitive to similar injuries, and yet everyone is agreed that the parietal peritoneum is highly sensitive. We determined to attack the problem from a somewhat different point of view. When a colotomy has been performed, the upper end of the gut opens freely upon the surface, and no fæces pass into the lower portion. It is therefore possible to wash out the lower gut, and by passing a tube into its upper end to apply heat and cold to what is now an isolated loop of intestine. By choosing patients who were intelligent, and such as were not cachectic or wasted from malignant disease, we obtained the following result. Water at 40° C. and at 20° C., which seemed warm and cold respectively to the skin of the abdomen, were entirely unappreciated when applied within the walls of the gut. But ice water was at once called "cold," water at 50° C. was said to be uncomfortable, and two patients of unusual intelligence spoke of this stimulus as "hot." This sensation of heat and cold was never localised in the abdominal cavity. If the patient was asked to indicate the position of the stimulus, he either placed his hand over the region of the navel or pointed into the air. Sometimes the sensation was said to be like cold drops on the skin in a part of the abdomen where it would have been impossible for any water to have fallen. Moreover, the strictest precautions were taken to insert the tube through a ring of mucous membrane and to surround both the tube and funnel with absorbent cotton-wool, so that no moisture could possibly escape.

These experiments are not conclusive, but they seem to show that, in some ways, the sensibility of the viscera closely resembles that which we have called protopathic. Only, the extremes of heat and cold are recognised, and localisation is so defective that the patient cannot even tell whether the cold is in or outside his abdomen.

Many of the afferent impulses from the viscera produce a reflex action without affecting consciousness. When we had passed a varying quantity of warm water into the gut, the patient complained that he wished to defæcate; a reflex peristalsis had been set up of which he was conscious, although he failed completely to recognise the stimulus by which it had been evoked. Most of the afferent impulses from the stomach and intestines probably belong to this order.

To a certain extent, we seem able to appreciate the muscular movements of an internal organ, such as the stomach or intestine, even although we cannot recognise the position in space of the part that is moved. This power is probably the equivalent of that deep sensibility which remains to a part deprived of all its cutaneous sensory nerves.

Structurally, we know that the viscera are innervated from the sympathetic system, and from a set of large afferent fibres connected with the end-organs of Pacini. The latter so closely resemble the mechanism found in muscles, tendons and joints, subserving what we have called deep sensibility, that we can assume the end-organs of Pacini to be the means by which we gain a similar power of appreciating intestinal movement.

But apart from such sensations of movement, the viscera certainly set up afferent impulses which may affect consciousness. We have attempted to show that, however feeble these sensations may be in consequence of the defective innervation of the intestine, they produce upon consciousness an effect resembling that of a low form of protopathic sensibility. Now, one of the peculiarities of protopathic sensibility is the rapid restoration of the mechanism upon which it is based. This it shares with the sympathetic system. Moreover, when a peripheral nerve to the hand is divided, it is noticeable that the palm begins again to sweat at a time after union which coincides approximately with that of the return of protopathic sensibility. This sweating is due to the motor fibres of the sympathetic (the "autonomic fibres" of Langley) that supply the skin.

It will therefore be no adventurous guess to suppose that the system we have called protopathic in the skin is one with the afferent fibres of the sympathetic as they supply the viscera. In both cases the sensation is badly localised, radiates widely, and is frequently referred to parts other than those stimulated. Both systems are incapable of appreciating light touch, and both are insensitive to the minor degrees of heat and cold. Both regenerate with the same rapidity and completeness.

We wish, therefore, to put forward a new conception of the nature of the afferent fibres in peripheral nerves.

The whole body within and without is supplied by the protopathic system. The fibres of this system in the skin may be spoken of as somatic, those to the internal organs as visceral protopathic fibres. Thus we shall no longer speak of the afferent sympathetic system, but of the protopathic supply of the internal organs.

Another set of afferent fibres peculiarly associated with impulses of movement and pressure exist in connection with the Pacinian organs. In the body and limbs, an analogous system is found peculiarly susceptible to pressure, to the localisation of movement, and to the appreciation of position. The fibres of this system run in conjunction with the motor nerves.

In addition to these two systems, which are distributed to all parts of the body within and without, the surface of the body only is supplied by a third system, which we have called epicritic. This endows the skin with sensibility to light touch. To the impulses conducted by this system we owe the power of localising the position of cutaneous stimuli, of discerning the doubleness of two points, and of discriminating between minor degrees of heat and cold, and other special attributes of sensation. The fibres of this system are more easily injured, and regenerate more slowly, than those of the protopathic system. They are evidently more highly developed, and approach more nearly, to the motor fibres that supply voluntary muscle, in the time required for their regeneration.

REFERENCES.

(1) SHERRINGTON. *Phil. Trans. Roy. Soc.*, 1896; *Medico-Chirurgical Trans.*, 1899, vol. lxxxii.
(2) BLIX. *Ztschr. f. Biol.*, 1885, Bd. xxi., S. 152.
(3) GOLDSCHEIDER. "Gesammelte Abhandlungen," Bd. 1, Leipzig, 1898.
(4) VON FREY. *Berichte d. König. Säch. Gesellschaft zu Leipzig. Math. physic. Classe*, 1894, ii., iii.; 1895, iii.; 1897, iv.
(5) LENNANDER. *Mitteil. aus d. Grenzgeb. d. Medizin u. Chirurgie*, 1902, Bd. 10, Heft 1 und 2.

THE CONSEQUENCES OF INJURY TO THE PERIPHERAL NERVES IN MAN.

BY HENRY HEAD, M.D., F.R.S.,
Physician to the London Hospital,

AND

JAMES SHERREN, F.R.C.S.,
Assistant Surgeon to the London Hospital; Surgeon to the Poplar Hospital for Accidents.

PREFACE.
CHAPTER 1.—NERVE SUPPLY OF THE PALM OF THE HAND.
§ 1.—Loss of Sensation after Division of the Ulnar Nerve.
§ 2.—Variation in the Extent of the Area supplied exclusively by the Ulnar Nerve.
§ 3.—Loss of Sensation after Division of the Ulnar Nerve when its Dorsal Branch remains Intact.
§ 4.—Loss of Sensation after Division of the Median Nerve.
§ 5.—Variation in the Extent of the Area supplied exclusively by the Median Nerve.
§ 6.—Loss of Sensation after Division of both Median and Ulnar Nerves.
CHAPTER 2.—RECOVERY OF SENSATION AFTER INJURY TO THE NERVES OF THE HAND.
§ 1.—General Statement of the Phenomena of Recovery.
§ 2.—Recovery after Division of Particular Nerves.
(*A*) Median Nerve.
(*B*) Ulnar Nerve.
(*C*) Median and Ulnar Nerves.
(*D*) Summary.
CHAPTER 3.—RECOVERY OF SENSATION AFTER INCOMPLETE DIVISION OF THE NERVES OF THE HAND.
CHAPTER 4.—NERVE SUPPLY OF THE FOREARM.
§ 1.—The Post-axial Half of the Forearm.
§ 2.—The Pre-axial Half of the Forearm.
CHAPTER 5.—INJURIES TO THE BRACHIAL PLEXUS.
CHAPTER 6.—LOSS OF SENSATION IN THE ARM FROM DIVISION OF POSTERIOR NERVE ROOTS.
CHAPTER 7.—NERVE SUPPLY OF THE LOWER LIMB.
§ 1.—The Sole of the Foot.
§ 2.—Loss of Sensation produced by Injury to the Nerves of the Leg.
§ 3.—The Nerve Supply of the Leg deduced from Residual Sensibility.

CHAPTER 8.—DEEP SENSIBILITY.
CHAPTER 9.—SENSATIONS OF HEAT AND COLD.
CHAPTER 10.—THE COMPASS TEST.
CHAPTER 11.—HAIR SENSIBILITY.
CHAPTER 12.—HYPERALGESIA.
CHAPTER 13.—CHANGES IN THE SKIN THAT FOLLOW INJURY TO PERIPHERAL NERVES.
CHAPTER 14.—CHANGES IN THE NAILS AFTER INJURY TO PERIPHERAL NERVES.
CHAPTER 15.—PARALYSIS AND OTHER MUSCULAR CHANGES.
CHAPTER 16.—THEORETICAL.

PREFACE.

GENERATIONS of anatomists have studied the course and distribution of the peripheral nerves, until knowledge of their more obvious features has apparently reached finality. It is recognised that more can be learnt of their central connections and of the relation of the larger branches to the anterior and posterior roots. But the peripheral distribution of the nerves of the hand is regarded as one of the commonplaces of anatomy.

And yet, whenever an attempt is made to apply this knowledge to some case where one of these nerves has been divided, obvious facts remain unexplained, or accessory hypotheses must be invented to account for the apparent difficulties of each individual instance. The more carefully the condition of the affected part is examined, the less does the state of its sensibility correspond with the surgeon's expectation. After he has successfully reunited the ends of the nerve, a careful examination only adds to the bewilderment of the conscientious observer.

If, for instance, the median nerve is divided, all cutaneous sensibility is abolished over a considerable part of both the index and middle fingers. But over the palm, within the area supplied by the median nerve, sensation may be diminished only. In a similar manner, division of the ulnar nerve produces complete insensibility of the little finger and of a variable portion of the ulnar border of the palm. Cutaneous sensibility is only partially lost over the palm and that part of the ring finger usually assigned to the ulnar nerve. When the surgeon or anatomist is asked why sensation is only partially lost, the usual answer is, "Because the nerves overlap." But, if each nerve occupies the territory of the other to an extent sufficient to prevent absolute loss of sensation over so large a part of the palm, it is obvious that destruction of the ulnar nerve must cause some diminution of sensibility within the median area. This loss should vary exactly in proportion to the amount of sensation that remains over this part of the palm, after the median nerve has been destroyed. But the most careful examination of the hand fails to reveal the slightest diminution of sensation over the median half of the palm, in consequence of division of the ulnar nerve. What has always been called diminished sensibility ends sharply at a line in the axis of the ring finger.

Such want of agreement between anticipated effects and the actual results of division of a peripheral nerve pointed to a gap in our knowledge of the distribution and functions of this part of the nervous system, which we have attempted to fill.

To those who have not worked in a town like London, it may seem an easy matter to examine a case of nerve injury at regular intervals from the date of the accident up to complete recovery. But any systematic attempt to carry out such an investigation is hampered by innumerable difficulties, due solely to the conditions of life among the working population of this huge city. Firstly, the original wound may have been treated at some other hospital, or by a private practitioner. Often the state of the wound and the extent of the injury can then be inferred only from the patient's description. Again, after the nerve has been successfully reunited, he may find it more convenient to attend some other hospital; or may leave his hand entirely untreated, and thus render useless the careful investigation at the time of the injury, the exploration of the wound at the time of suture, and the observations made during his stay in hospital.

Lastly, the investigation may be brought to a sudden end by his change of dwelling. For instance, within the space of twelve months, one of our cases, a married man with a family, changed his address five times. Two or three changes in a year are of frequent occurrence, and letters remain unforwarded. In spite of the help of an assistant, skilled in tracing the movements of hospital patients, and in spite of the fact that compensation on an ample scale was given for travelling expenses and loss of time, many cases disappeared entirely, often at the most interesting period of recovery. This is particularly liable to occur when the median has been divided. For this injury interferes little with the grasp of the hand, and the patient is afraid to attend the hospital, lest his employer should consider him unfit for work. To meet this difficulty, we found it necessary to institute frequent Sunday sittings.

No cases are included in this paper that have not been examined by one or both of us. As far as possible, one or other of us has been present during the operation; but occasionally we have been compelled to rely on the account given by others of the condition then found. With this exception, no note has been included that is not the direct outcome of our personal observation.

Cases of nerve injury usually remain but a short time in hospital, and all the later treatment is conducted in the outpatient department. But in every instance, the observations we record were made under conditions of time and space more favourable to accurate investigation. Certain mornings in the week were specially set aside for the work, and the patients were seen, one by one, in a quiet room.

First of all, the patient was asked to give an account of the condition of the part affected and any change that he might have noticed since his last visit. The state of the skin was noted and the growth of the nails measured. The movements of the injured limb were investigated; but its electrical reactions were not tested until the end of the sitting.

During the testing of sensation, the patient's eyes were closed, and, if necessary, bandaged; he was told to call out whenever he experienced a touch, a prick, or any other sensation. No further questions were asked.

Superficial touch was tested by means of cotton-wool stroked gently across the palm. Unfortunately, this stimulus, so perfect a test over parts endowed with hair, is untrustworthy over hairless parts, unless used with extreme caution.[1] Many brands of wool, when rolled into a whisp, form so stiff a mass that deep sensibility is evoked. The untreated cotton-wool used by jewellers is excellent for our purpose. But the cotton-wool of short staple, which has been rendered absorbent by the action of potash, is too stiff to be a certain stimulus for superficial touch alone. Moreover, certain parts, even of a well-kept hand, such as the outer aspect of the thenar eminence, are relatively insensitive to superficial stimulation with cotton-wool, and over the greater part of the horny hand of a workman this stimulus may evoke no response. Cotton-wool must, for these reasons, be used with extreme care, and in every case the condition of the skin must be noted with accuracy. But we wish to call attention to another source of error produced by the roughness of a desquamating skin. Cotton-wool brushed lightly across a part in this condition may be appreciated through that form of deep sensibility, which remains intact after all cutaneous nerves have been destroyed. It would then appear as if light touch were appreciated, whereas, in reality, the sensation evoked is that of pressure.

The power of discriminating two points was tested with a pair of compasses, the ends of which had been blunted. But the method adopted differs from that in general use, and requires closer description (*vide* also p. 228). The compass points were set at a certain distance from one another; they were then applied to the part to be tested in such a way that sometimes two points, sometimes one point only, touched the skin. No questions were asked, but, before the testing began, the patient, whose eyes were closed, was told to say with every application how many points he had perceived. The stimuli followed one another in an entirely irregular order, but so that, ultimately, the patient had been touched ten times with one point, ten times with two points. Each series was recorded in such a manner that it became immediately obvious in how many cases, among the ten single and among the ten double stimuli, the patient had given a wrong answer. We look upon this method of recording the result of single applications interspersed among an equal number of double ones as a great addition to the practical value of this test, for, when the threshold is neared, one of the first changes is a tendency on the part of the patient to become uncertain about the nature of single stimuli. At this particular distance, every touch, whether single or double, is liable to be called two. Thus, the very appearance of this condition, usually supposed to show that the patient is untrustworthy and the observations worthless, is in itself one of the most valuable signs that the threshold for the discrimination of two points is about to be reached.

---

[1] During the recovery of sensibility hair-clad parts may react in a peculiar manner (*vide* Chap. 11).

The sensation of pressure was tested by means of a pencil or some other blunt object. Care was taken to record the locality to which the stimulus was referred, verbally or by indication.

The affected parts were also tested with a tuning-fork, and for this purpose we have usually used one beating 128 double vibrations a second. This is a treacherous form of stimulation, unless the patient is intelligent and the observer constantly on his guard. Error not uncommonly arises in consequence of conduction of the vibrations from an insensitive to some sensitive part at a distance; the patient then appreciates the stimulus, but not at the spot to which the fork was applied.

Wherever it was necessary to investigate the patient's ability to appreciate the position into which a joint had been passively placed, the test was carried out as follows: He was told that the fingers affected would be moved, and that he was to imitate with the sound hand the position into which they had been placed. When the sense of passive position is perfect, an intelligent patient can carry out this movement of imitation with remarkable accuracy.

A sharp needle or a steel pin was used as the test for pain, and care was taken to differentiate between the sense of deep pressure so produced and true pain. If there was any doubt as to the sensation experienced, a painful interrupted current was used as the test, and by this means it was easy to discover whether the patient's answers were based upon deep pressure or upon the true appreciation of pain.

When sensibility to pain has returned, the limits of the loss of the higher forms of sensation can be discovered by the following method: The point of a needle or a steel pin is dragged lightly across the skin, from normal parts towards the affected area; immediately its borders are reached the patient cries out, saying that the discomfort is greatly increased. This will be spoken of as "the line of change to prick."

Many difficulties surround the testing of sensibility to heat and cold, particularly as minor degrees of temperature play so considerable a part in our investigation. An ordinary glass test tube containing ice has been used for cold, and serves this purpose admirably. For the sake of convenience, under the somewhat difficult conditions of clinical research, we have also used glass tubes in testing for heat, for warm, and for cool. But, used for this purpose, a glass tube is open to serious objections, for, if water is heated to a temperature of 50° C., the glass wall of the tube will be considerably hotter than the contained water. Thus, a thermometer placed in the water does not register the actual temperature applied to the patient's skin. In a similar manner, it is long before hot water poured into a glass tube raises the external temperature to that of the contents, and heat abstracted from the wall of the tube by contact with the skin is only restored by slow degrees. The following precautions must be observed if glass tubes are used: The tube must be over-heated and put aside to cool to the point desired; repeated stimuli must not be made at short intervals with the same test tube, and, for this reason, it is well to have a row of tubes for use as they reach the temperature required. In some of our more delicate observations we have used silver tubes; with careful use they obviate all the difficulties inherent in glass test tubes.

We cannot close this description of the manner in which our work has been conducted, without expressing our sincere thanks to all those who have generously permitted us to make use of the patients under their care.

Without the cordial help of the surgical staff of the London Hospital, and of the Poplar Hospital for Accidents, this work would have been impossible.

Sir Victor Horsley has alone enabled us to complete one section of this work by the liberality with which he allowed us to examine patients, in whom he had divided the posterior roots.

To Surgeon-General Stevenson, C.B., and the surgeons in charge of the Royal Victoria Hospital, Netley, we return our thanks for permission to make use of patients injured during the South African War.

Our thanks are also due to Professor A. E. Barker for leave to examine and to publish an important case under his care at University College Hospital.

Lastly, we return our thanks to the Surgical Registrars, past and present, of the London Hospital (Mr. Lett and Mr. Russell Howard), for unfailing help in the collection of patients, who attended the hospital for nerve injuries.

CHAPTER 1.—NERVE SUPPLY OF THE PALM OF THE HAND.[1]

§ 1.—*Division of the Ulnar Nerve.*

Complete division of the ulnar nerve in the forearm above the point at which the dorsal branch is given off produces the following changes in the sensibility of the hand.

Touch, prick, heat and cold are no longer appreciated over the little finger and over the ulnar border of the palm. The extent of this absolute loss of cutaneous sensation varies in each individual, and in no two cases is it exactly the same. In one extreme form it may occupy the little finger, one-half of the ring finger and more than one-third of the palm and dorsum of the hand, or sensation may only be completely lost over the little finger and ulnar border of the palm.

When the whole ulnar nerve is divided, the area of absolute loss of cutaneous sensation lies between these two extreme limits, the amount of loss varying with each individual. And it is this loss of sensation only which can be recognised by pricking the hand with a pin. If one finger and a half are insensitive to a prick, the surgeon is satisfied that he has to deal with a "normal" ulnar "completely divided"; when only the little finger is insensitive, he doubts whether the nerve is completely divided, or looks upon it as abnormally distributed.

---

[1] For the extent of the nerve supply of the hand determined by the method of residual sensibility, *vide* p. 184.

But in reality, the sensibility of the hand is disturbed over an area considerably greater than that marked out by the analgesia; and, if cotton-wool is used as the test for sensation, touches, easily felt elsewhere on the hand, will not be appreciated over the whole of that portion assigned by anatomy to the supply of the ulnar nerve. This area is bounded by a line running through the longitudinal axis of the ring finger back and front, continued on the dorsal and palmar aspects of the hand to include the greater part of its ulnar half.

The whole of this border can be marked out easily with cotton-wool, for, as soon as it is passed, the patient appreciates touches that previously caused no sensation, and, if he is intelligent and quick, the passage from the insensitive to the sensitive area is found to take place at a line which varies very little, whether the stimuli progress is an orderly series from the ulnar to the radial side of the hand, or *vice versâ*.

Thus, by using cotton-wool as a test, sensation can be shown to be lost at a line corresponding to the anatomical border of the ulnar nerve.

But there are other means of showing that sensation becomes defective at this border. If a needle or pin is dragged lightly across the skin from the sound to the affected half of the hand, the patient complains that the "feeling" it produces changes as soon as this line is passed. This line can also be marked out by an interrupted current applied in the following manner: Connect one pole of the secondary coil with a large indifferent electrode, and the other pole with a small electrode covered with wash-leather set in a handle containing a key, so that the current can be thrown in and out at will. Remove the iron core from the primary coil, then place the secondary coil at such a distance from the primary, that the current applied through the smaller electrode is easily appreciated on the normal skin. Even though it may be strong enough to contract the small muscles of the thumb, such a current will not be appreciated over the area within which sensation is lost to cotton-wool.

Sensation to temperature also undergoes a change at the same border. A test tube containing water at about 22° C., and one containing water at about 40° C., cannot be discriminated, though easily appreciated as cool or warm over the normal skin.

Thus, division of the ulnar nerve produces complete loss of sensation to pain and temperature over the little finger and over a variable extent of the palmar and dorsal surfaces of the ulnar border of the hand, rarely corresponding even approximately to the anatomical borders of the ulnar nerve. But these borders are accurately marked out by loss of light touch (cotton-wool), and of minor degrees of temperature and by inability to appreciate the interrupted current applied in a definite way.

Between the boundaries of this loss of light touch and, those of complete loss of cutaneous sensibility lies a territory within which sensation is profoundly changed. The extent of this area varies in each individual case. If the intermediate zone be of considerable size, so that the condition of sensation within it is easily investigated, then it will be found that not only is all sensation abolished to light touch, to intermediate degrees of temperature, and to a certain form of interrupted current, but painful stimuli produce an effect different from that upon the normal skin. As soon as the anatomical border is transgressed towards the ulnar side, a prick may become so disagreeable that the patient immediately withdraws his hand. He complains that it causes a feeling of "pins and needles," not only at the point pricked, but also widely over the intermediate zone. Asked to localise the spot pricked, he may be able to do so, but complains that the pain produced seems to him to be spread over a large surface, or even to be in two places at once, such as the base of the finger and the middle of the palm. Moreover, when tested with compasses, the points cannot be distinguished as causing two sensations, even when separated from one another to the greatest extent possible within the intermediate zone. An interrupted induced current, with no iron in the circuit, cannot be appreciated within this area, but, if bare metal points are used, or if the iron core is inserted into the primary coil, the stimulus causes pain, even when the distance of the coils is adjusted to compensate for the increase of strength produced by the presence of the core.

Thus, complete division of the ulnar nerve above the dorsal branch produces the following changes:—

(1) Loss of sensation to pain, to extremes of heat and cold (ice and water at 50° C.), and to painful interrupted induced currents, over an area that may vary greatly in size; sometimes it includes the little finger, the ulnar half of the ring finger, and more than one-third of the palm and dorsal surface; in other cases it is reduced to the little finger and a strip on the extreme ulnar border of the hand.

(2) The patient is unable to distinguish two widely separated compass points, or to appreciate light touch, minor degrees of temperature, and the painless interrupted current over an area occupying the little and ulnar half of the ring fingers, and that part of the palm and dorsum of the hand on the ulnar side of an axial line drawn longitudinally through the ring finger. This corresponds to the border laid down by anatomy for the supply of the ulnar nerve.

(3) The sensibility of the intermediate zone on the palm, the dorsum and the ring finger may be characterised by an increase in the discomfort produced by painful cutaneous stimuli, and by a wide diffusion and want of localisation in the sensation produced by a prick.

§ 2.—*Variation in the Extent of the Area supplied by the Ulnar Nerve.*

Complete division of the ulnar nerve produces loss of sensation to light touch over the whole of the little finger

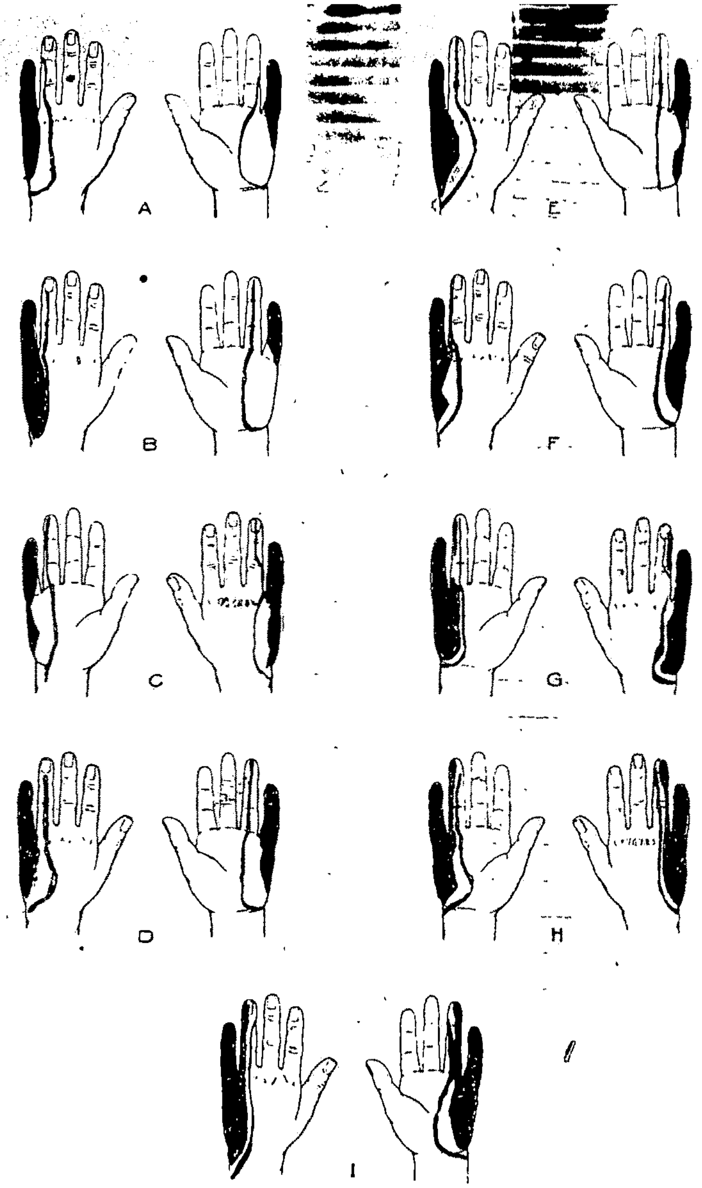


Fig. 1.

To show the loss of sensation produced by complete division of the ulnar nerve. Loss of all forms of cutaneous sensibility is represented by the black area. The parts insensitive to light touch and to the intermediate degrees of heat and cold are enclosed within the black line.

Most of these cases will be found on Table II., p. 156.

A is the loss of sensation in Case 18; B, Case 17; E, Case 16; F, Case 14; G, Case 15; I, Case 19 (also reported in full in the Appendix, p. 309). The case from which C was taken (No. 83) will be found reported on p. 311; that where the loss of sensation was represented by H, on p. 316 (No. 63). D was taken from a man in whom the ulnar nerve had been divided; secondary suture was performed, and he was not seen again until complete recovery had occurred.

and over some part of the ring finger back and front. In no instance was the sensibility of the ring finger to light touch entirely unaffected. Of nine cases in which the nerve was proved at the operation to have been divided, this anæsthesia occupied the ulnar half of the ring finger in six, and this may therefore be taken to represent the usual supply.

On the palm, the loss of sensation to light touch may occupy a border directly continuous with the axis of the ring finger, or may swing out as far as a line drawn from the cleft between the middle and ring fingers (fig. 1).

Out of these nine cases loss of light touch occupied half the dorsal surface of the ring finger in six; in one, a third of this finger was affected. In one instance, this anæsthesia occupied two-thirds of the ring finger.

On the back of the hand, the border of the area insensitive to light touch usually follows a line continuous with the axis of the ring finger, but in two cases it swung out to the radial side to reach the tendon of the middle finger.[1]

The ulnar nerve is usually divided by a transverse cut in the neighbourhood of the wrist, which must also sever some of the branches of the internal cutaneous nerve descending to supply the hand. Thus, in most cases, the scar bounds the upper or central border of loss to light touch, and it is only where the ulnar nerve has been injured at the elbow, or high in the forearm, that the true upper limit of the ulnar supply can be determined. Six such cases have come under our notice, in four of which this upper limit ran round the wrist at the level of the styloid process, and in two it formed a curved line about 1 cm. on the distal side of this point. Evidently, as far as light touch is concerned, division of fibres of the internal cutaneous nerve plays little part in the form usually assumed by this border after division of the ulnar nerve at the wrist, excepting when the anæsthesia is bounded definitely by the scar.

Thus, the extent of the loss of light touch produced by division of the ulnar nerve seems to be remarkably constant, and it varies, if at all, only within small limits. The borders of this area are definite; it does not merge gradually, but passes abruptly into parts of normal sensibility.

---

[1] Under certain circumstances the presence of hairs on the dorsal surface of the hand may make the determination of this border untrustworthy unless the hand be shaved (*vide* p. 239).

The area of insensibility to pain, produced by division of the ulnar nerve, differs fundamentally in every characteristic from the condition of parts insensitive to light touch. For, not only is the extent of the loss of sensation subject to great variation, but the difficulty in determining the amount of this variation is increased by the indefinite nature of the borders of the analgesic area. At no point can it be said that here loss to pain begins; complete sensibility to pain merges gradually into complete insensibility with no sharp dividing line, and all attempts to mark out circumscribed areas of analgesia are therefore unsatisfactory. But, taking into account solely the area of total loss of sensation to prick, the only certain result of complete division of the ulnar nerve is to produce analgesia over the little finger and ulnar border of the hand. None of our cases failed to show at least so much loss to prick. But, in extreme instances, sensation to prick may also be lost over the ulnar half of the ring finger and over an area on the palm and dorsum of the hand almost co-terminous with the full ulnar loss of sensation to light touch. It is in such cases that the surgeon, using a pin as his test for sensibility, finds that loss of sensation occupies exactly the area he expected.

Between these two extremes, every form of variation exists; in no two cases is the extent of the complete analgesia exactly the same, and so diverse are the forms assumed by this loss of sensation that no form can be said even approximately to represent the normal. We have therefore represented the extent of the loss, in each case, in the form of a series of diagrams, from which it will be seen how great may be the variation (fig. 1).

Apparently, the extent of the area insensitive to light touch, and that of the area of absolute loss of sensation to prick, vary independently of one another. A large extent of the ulnar half of the hand may be entirely insensitive to pain, and yet the extent of loss to light touch in xceeds that found when the analgesia was confined tle finger.

For this reason, the extent of the intervening zone of defective sensibility varies greatly. Its characteristics are an imperfect discrimination of two compass points and an inability to transmit light touch and degrees of temperature between about 22° C. and 40° C. It is, however, sensitive to pain, to ice, and to temperatures above 45° C. But, in consequence of the ill-defined borders of this total loss of sensation, the intermediate zone may sometimes be an area of very defective sensibility, or it may be sufficiently large and sensitive for careful and certain examination of its sensory peculiarities.

§ 3.—*Loss of Sensation produced by Division of the Ulnar Nerve, when its Dorsal Branch remains Intact.*

When the ulnar nerve is divided at the wrist, its dorsal branch not uncommonly escapes uninjured. Such an accident makes it possible to determine the extent to which each of the two branches supplies the ulnar area of the hand.

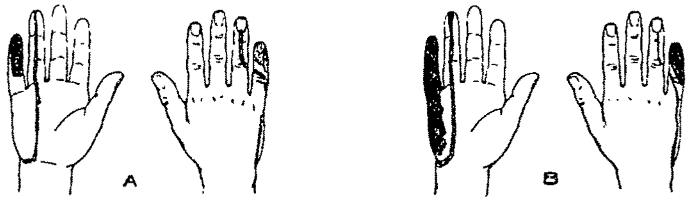


FIG. 2.

To show the loss of sensation produced by division of the ulnar nerve below its dorsal branch. The area of total loss of cutaneous sensibility is marked in black. The parts insensitive to light touch and to the intermediate degrees of heat and cold are enclosed within a black line.

Both these cases will be found on Table II., p. 158. A represents the loss of sensation in Case 20; B the loss of sensation which preceded and immediately followed secondary suture in Case 24.

When the dorsal branch is intact, the border of loss to light touch coincides on the palm with that found after complete division of the ulnar nerve. The whole palmar surface of the little finger and the greater part of the ulnar half of the [illegible]ger are insensitive to cotton-wool. On the dorsu[illegible] hand, the loss of sensation may occupy

the ulnar half of the two terminal of the ring,
and the whole of the two terminal s of the little
finger; or the whole little finger an portion of the
ulnar border of the dorsal surface hand may be
insensitive to light touch. But, whe ay be situated
in any individual case, the border the loss of
sensation on the palm, from the no on the back
of the hand, is an indefinite one. ly, whenever
loss of light touch has been under , the borders
of such loss have been spoken of as hat is to say,
the passage, from a part over whic wool is appre-
ciated to one where it no longer p ny sensation,
is so rapid, that for practical purpose e represented
by a line. This is not the case w orsal branch
of the ulnar nerve has remained inta ulnar portion
of the palm of the hand is insensitiv n-wool; but,
as the stimulus progresses towards surface, the
point at which it first evokes a sens ncertain, and
the area of anæsthesia seems to me ally into the
complete sensibility of the back of th

The extent to which sensation and to the
extremes of heat and cold is lost vary greatly.
It may be that the only absolute lo tion is found
over the terminal two phalanges of finger on the
palmar aspect, and over the termin behind; or
the whole ulnar third of the palm, th lmar surface
of the little finger and its two term anges on the
dorsal surface may be entirely insens

This absolute loss of sensation, ho ensive it may
be, merges gradually into the area o ss and is not
constant. Like all parts where sen prick and to
the extremes of heat and cold is def extent of the
loss varies according to the temperatu hand and the
general condition of the patient. Th 24 (Table II.,
fig. 2, B, p. 130), the sensibility impr gain deterio-
rated, although the two ends of the rve remained
effectively separated.

In most cases, where the dorsal br tact, the con-
siderable extent to which the borders light touch

and to prick are separated from one another, renders it particularly easy to determine the character of sensation obtained from the intermediate zone. When pricked, the patient withdraws his hand with an exclamation, as soon as the area is reached where light touch is lost. Ice and water at 50° C. can be appreciated, water between about 22° and 40° cause a sensation of touch only. But, even though a prick can be perceived, it produces a widely diffused, tingling sensation, and two compass points are not distinguished, even when separated for a distance of 4 cm.

The dorsal branch seems to supply sensibility to light touch to the lower half of the ring finger on its ulnar aspect, and to the greater part of the ulnar third of the back of the hand. If it remains intact, there may be no loss of sensation to prick on the palm or first and second phalanges of the little finger after division of the ulnar nerve. Or the extent of the area supplied by the dorsal branch may be so small that all sensation is lost over the ulnar palm, and the analgesia occupies the two terminal phalanges of the little finger, and even laps slightly on to the dorsal surface of the hand.

Thus, it would seem that the part played by the two main branches of the ulnar nerve in supplying sensation of light touch to the hand varies little, but the border between the areas they supply, unlike any touch border yet described, is not fixed; the parts, where sensation is lost to light touch, merge gradually into the back of the hand, where sensation is unaffected. But there is great individual variation in the extent to which sensation to prick and to the profounder degrees of heat and cold is lost. No two cases are exactly alike, and any focus of absolute analgesia that exists is surrounded by a wide area of partial loss to prick.

§ 4.—*Division of the Median Nerve.*

Usually, when the median nerve is divided, the skin over the dorsal and palmar surfaces of the two terminal phalanges of the middle and index fingers becomes insensitive to light touch, pain and temperature. But cutaneous sensibility

may also be lost over a wider area. nar aspect of
the thumb, the hypothenar eminenc greater part
of the median half of the palm may b ly insensitive
to prick. In such cases, the loss of of cutaneous
sensation nearly corresponds to the ar d by anatomy
to the supply of the median nerve. B t a loss is not
present in the majority of cases. Th to which the
palm and the palmar aspect of the th ffected varies
greatly; but on the dorsal surface of and middle
fingers, the boundaries of the anal remarkably
constant, reaching as a rule to the f e skin over
the first interphalangeal joint of both

The extent to which sensation to to tempera-
ture is completely lost varies greatly t be said to
be exactly similar in any two cases. e area over
which light touch cannot be apprecia re constant.
Its borders usually extend from the rad the thumb-
nail along the radial border of the th e fold at the
base of the thenar eminence; thence up the great
central line of the palm to the cleft be middle and
ring fingers. It includes a variable por e radial half
of the palmar surface of the ring fing the dorsal
surface the radial third of the termi halanges of
the ring finger, and the skin over th two and a
half phalanges of the middle and ind From the
radial side of the index finger the bord towards the
thumb, running along the extreme fr of the first
interosseous space, and thence extends umb, to end
at the ulnar border of the nail. The which light
touch is lost, corresponds on the palm xactly with
that assigned by anatomy to the media

Although cotton-wool is the best m arking out
this border, light touch is not the onl sensation
which there undergoes a change. Te es between
about 22° C. and 40° C. are entirely un as soon as
this line is passed, and the painless d current,
generated with no iron in the circuit, this border
to cause sensation, though well apprec he normal
skin.

Thus, after division of the median nerve, exactly as with the ulnar nerve, an intermediate zone makes its appearance between the boundary for loss of light touch and the boundary of those parts over which sensation is absent to prick. Closer examination of this intermediate zone shows that sensation, produced by stimuli applied within it, has the same characteristics as that from the similar area, caused by division of the ulnar nerve. A prick usually causes pain more disagreeable in character than that of the normal skin. The patient has an urgent desire to withdraw his hand and cries out, or shows some obvious sign of discomfort. The sensation produced is widely diffused and badly localised; it is said to be a "numb-tingling pain." So characteristic may be this form of sensation, that the border at which light touch ceases to be perceived can be frequently marked out by dragging the point of a pin lightly across the skin from normal to abnormal parts, noting at what point the character of the sensation so produced undergoes a change.

Temperatures between about 22° C. and 40° C. are unperceived when applied within this area; but the more extreme degrees of cold and of heat are usually well appreciated, although they cause a diffuse, badly localised, tingling sensation, unlike any effect produced upon the normal skin.

§ 5.—*Variation in the Extent of the Area supplied by the Median Nerve.*

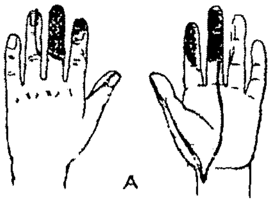
Among the twelve cases, where the median nerve proved at the operation to have been divided, there was but little variation in the extent of loss of sensation to light touch and minor degrees of temperature. In all, the ring finger was affected to a greater or less degree, usually one-half (six cases) or one-third (three cases) being anæsthetic; but in two instances, the anæsthesia occupied a small portion only of its extreme radial aspect, and in one two-thirds of the whole finger. On the palm, the border may vary between a line drawn through the axis of the middle, and one drawn through the axis of the ring finger. The small variations which occur on the dorsal surface, in the extent to which the index and middle fin isensitive, can be best appreciated from the series ms on fig. 3. The greatest variation occurs on t finger, where the anæsthesia may extend over the nal phalanges or may occupy the whole finger t . Thus, the extent of the loss of sensation to lig and to minor degrees of temperature, is remarka nt and varies within small limits.

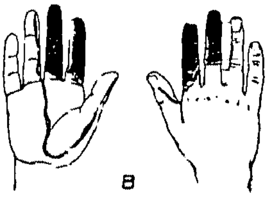
These differences are trivial co ith the wide variations in the extent of the loss tion to prick, variations so profound that no two can be said to resemble one another exactly. I ng the extent of this analgesia, it is important to nly in which the nerve was proved by operation to n divided, and to choose only such observations as le as soon as possible after the occurrence of the fore recovery could have begun.

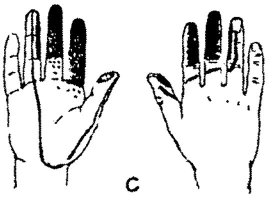
Twelve of our cases come up to ard. Among them five showed so large an amount f sensation to prick, that the whole of the palm signed to the median nerve, together with the palm of the thumb and both index and middle fingers, wa . In fig. 3, L, the loss of sensation to prick on th as somewhat less extensive, but one-third of the ri as analgesic. In every case, the extent of this loss of was different, until in fig. 3, A, it reached the smalle ions we have yet seen. Here, scarcely the termina langes of the index and middle fingers were affect ick could be appreciated over the whole of the pal nd and over the palmar aspect of the thumb.

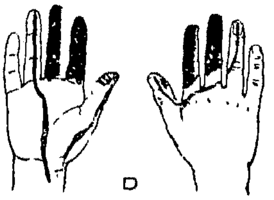
Thus, it is impossible to lay down al rule, even with regard to the usual extent and on of loss of sensation to prick when the median een divided. We can only say, that, when it reach est extent, it almost corresponded to the area of lo tion to light touch, or, when the analgesia was r its smallest proportions, scarcely the whole of the t vo phalanges of the index and middle fingers wer l insensitive to prick. Between these two extrem variety may occur.

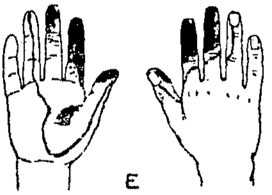
It might be supposed, that the presence of a considerable area of loss of sensation to prick on the palm depended upon injury to the descending branches of the external cutaneous

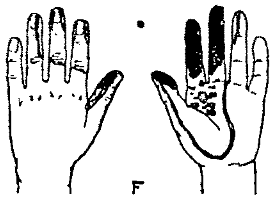

A

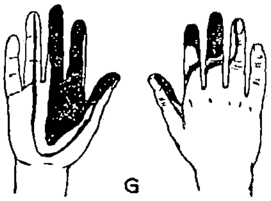

B

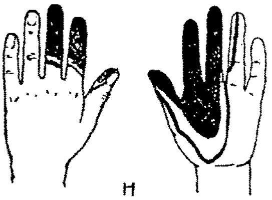

C

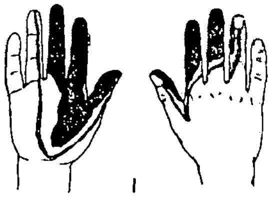

D

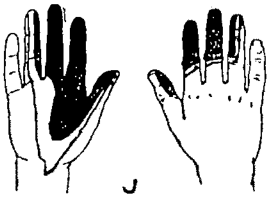

E


F


G


H


I


J

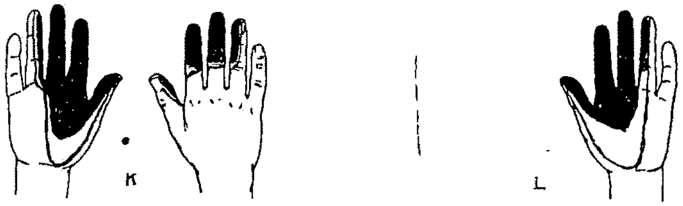


FIG. 3.

To show the loss of sensation produced b
he median nerve.
The area of complete cutaneous insensibility i
black. The parts
insensitive to light touch and to the intermedi
f temperature are
enclosed within a line.

Most of these cases will be found on Table

A represents the loss of sensation in Cas
11; C, Case 13;
D, Case 6; E, Case 3; F, Case 12; G, Ca
se 4; I, Case 9;
J, Case 10; L, Case 5. K is taken from a wom
letely divided her
median nerve in the neighbourhood of the
examined her on
several occasions, but she disappeared before re
sation began.

nerve. But in the patient from w
K, was taken
the nerve was divided by a wound i
of the elbow.
Moreover, in fig. 3, L, the extent of
sia was larger
than in any other instance that has
er our notice,
and yet the nerve had been divided
small punc-
tured wound at the wrist, which coul
e injured any
considerable number of fibres of the
itaneous.

§ 6.—*Division of both Median* *Nerves.*

An extensive wound of the wrist
de both the
median and the ulnar nerves, causin
is of all the
intrinsic muscles of the hand and wi
oss of sensa-
tion. To produce such great destru
wound must
be unusually severe, and, commonly
other nerve,
though injured, escapes complete d
This is the
condition in the majority of those ca
the median
and ulnar nerves are supposed to ha
ivided. But
amongst our patients were two in w
nerves were
seen to be cut across at the time of t
wound, and
one, where they were divided seven w
the original
injury for the purpose of secondary su

Taken in connection with a numb
where both
nerves were gravely injured, this mat
gh small, is
sufficient to determine the extent to
ht touch is

affected when the two nerves are completely divided. But the loss of sensation to prick varied so greatly in the three instances of undoubted division, that it is impossible to say to what extent this form of sensation is most commonly lost.

Sensation to light touch is abolished by this injury over the whole palm and over the palmar aspect of the thumb and all the fingers The outline of this area on the thumb corresponds, when uncomplicated by injury of other branches, to the similar border produced by division of the median nerve, and, like it, varies in the extent to which the thenar eminence is involved. Sometimes the anæsthesia over the proximal part of the base of the thumb is too extensive to be due entirely to destruction of the median nerve, and is probably caused by division of fibres from the external cutaneous descending on to the palm. Any cut, running across the wrist from side to side completely, must tend to divide these branches.

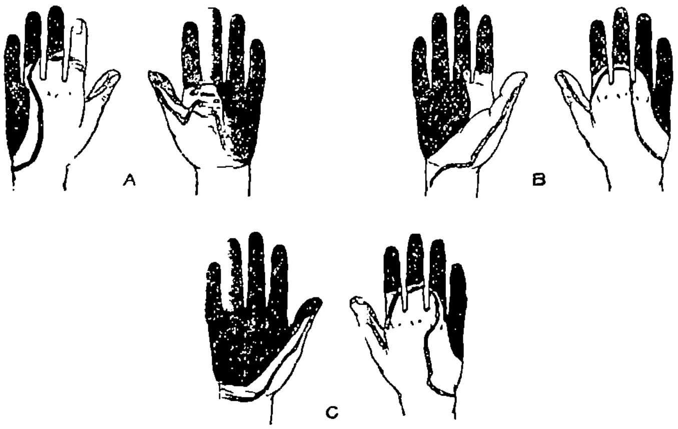


FIG. 4.

To show the loss of sensation produced by complete division of both median and ulnar nerves. The area of complete cutaneous insensibility is marked in black. The parts insensitive to light touch and to the intermediate degrees of temperature are enclosed within a line.

These cases will be found on Table III., p. 160.

A shows the loss of sensation in Case 26, B in Case 28, and C in Case 25.

On the posterior surface, the border of the area over which light touch and minor degrees of temperature are lost varies, according to whether th ranch of the ulnar nerve has been severed or not. hree cases of complete division of both main trun ar nerve had been divided above the point at whic ch was given off. The loss of sensation on the bac nd, therefore, corresponded to that seen after compl n of the ulnar nerve. On the dorsal surface of the in iddle fingers, the anæsthesia extended to the prox over the first interphalangeal joint in two cases, a oint half-way between this fold and the knuckle i rd. On the thumb, the border ran from the ulna the base of the nail to the dorsal aspect of the free e first inter-osseous space. Thence it passed up t aspect of the base of the index to join the line rsum of this finger.

The extent to which light touch inor degrees of temperature were lost corresponde to the loss of sensation produced by division of the ve, added to that caused by division of the media sionally, the loss on the palmar aspect of the thum tle increased by destruction of branches of the exter ous running downwards over the wrist.

To prick, the loss of sensation vari tly that an attempt to describe in detail its bo n each case would be wearisome, and the reader d to fig. 4. The greatest loss appeared in Case 2 , where the whole palm and palmar aspect of the t e insensitive to prick. But it must be remembered his patient, all the structures on the front of the w een divided to the bone, and amongst them must h ncluded the descending branches of the external nerve. In both the other cases, loss of sensati k was less extensive on the palm (fig. 4), and th ly belonged to the group in which the median nerv exclusively the fingers only.

On the dorsal surface, the index, m ring fingers were insensitive from the tip to the ld over the first interphalangeal joint in two of th in one, the dorsum of the index seemed to be sensi ck. In all, the whole of the little finger and a varying portion of the ulnar aspect of the dorsum of the hand were analgesic. The material at our disposal is small; but it would seem that division of the median and ulnar nerves tends to produce the following results:—

(1) Sensation to light touch is lost over the whole of the palm. Loss of sensation on the back of the fingers extends at least to the first interphalangeal joint; and if the ulnar nerve has been divided above its dorsal branch, the anæsthesia invades the whole of the ulnar half of the middle finger, the whole little finger, and a variable extent of the dorsal surface of the hand.

(2) The loss of sensation to prick varies greatly in extent. In one instance, the whole palm was insensitive to prick. In the remainder, the thenar eminence and the extreme radial portion of the hand were sensitive to this form of stimulation. On the dorsal surface, the index, middle, ring, and little fingers became analgesic over an area which varied in each case (fig. 4).

CHAPTER 2.—RECOVERY OF SENSATION AFTER DIVISION OF THE NERVES OF THE HAND.

§ 1.—*General Statement of the Phenomena of Recovery.*

The ultimate consequences of division of one of the nerves of the hand depend entirely upon the treatment adopted. If the nerve be sutured, and the wound heal by first intention, sensation may return to a condition indistinguishable from that of the normal skin. And in the progress of such return, the hand will pass through stages that throw much light on the structure and functions of peripheral nerves.

Division of the nerve leads at once to the production of an area of absolute cutaneous insensibility, surrounded by an area of loss of sensation to stimuli, such as light touch and the minor degrees of temperature. The relative extent of these two areas differs greatly in each individual case, and the first definite sign of recovery is shown by an increase in size of the intermediate zone between them. At the end of a variable period after division of the nerve, the analgesia

begins to retreat from the palm of and occupies
the fingers only. Gradually, it passe igers joint by
joint, until at last there is no part nd or of the
fingers, where prick cannot be ap During the
whole of this period, the area o light touch
remains as well defined as on the d accident; it
takes no part in the recovery of sen d yet, at the
end of several months, the whole of of the hand
supplied by the affected nerve has bec ive to painful
stimulation.

Even pressure with blunt objects, pencil or the
head of a pin, causes pain, and the mplains that
an accidental knock over this part of is extremely
unpleasant. In quality, the sensation affected half
of the hand resembles that of the int zone, which
was found between the border for ligh d the border
for prick shortly after the accident. h is entirely
unperceived, and two points of the s cannot be
discriminated, even when widely separ prick causes
a diffused sensation of "pins and ne "tingling,"
which is localised, not only at the poin ation of the
stimulus, but widely over the affected e hand. A
test tube containing water at any tem elow 20° C.
is appreciated as cold, and it matters li er it contain
ice or water at 18° C., both are said to ld." Water
at 50° C. causes a stinging, correspon unpleasant
aspect of the sensation produced on th hand by too
hot water. This may or may not be a d by a true
sensation of heat, according to the sta ery reached
by the affected hand. But whether tr e present or
not, patients usually speak of the "st roduced by
water at 50° C. as "hot" or "burni se no other
common natural stimulus is capable of is peculiar,
unpleasant sensation.

In the earlier period of recovery, w nalgesia is
retreating from the hand, all sensation eat is, not
infrequently, absent. The recovering sensitive to
cold and to the unpleasant "burning" ng" aspect
of a hot stimulus, but not to heat itself. ons of cold

play, therefore, a greater part in the effect produced by this area of the skin upon consciousness, and this part of the hand always "feels colder" than normal. Ultimately, however, temperatures of 50° C., or above, can be appreciated without hesitation as heat. Yet, throughout this stage of recovery so long as light touch is completely absent over the affected area, water at 40° C. and below produces no sensation of warmth; it cannot be distinguished from water at 25° C., and is said to be neither hot nor cold.

Thus, when the whole portion of the hand affected has become sensitive to prick, the sensations evoked closely resemble those arising from the intermediate zone in their diffuseness and want of strict localisation, and in the fact that degrees of temperature between about 25° C. and 40° C. cannot be appreciated.

But although sensation from the whole affected parts of the hand closely resembles in quality that of the intermediate zone present immediately after the nerve has been divided yet, in intensity and rapidity of reaction, the sensitiveness of the recovering parts is considerably greater. We have no satisfactory measure of the intensity of pain, and can judge only by the statements and behaviour of the patient. By such standards it is certain, that a prick now produces a more unpleasant sensation over the same parts than shortly after the accident before recovery could have begun. Moreover, cold and heat are felt with greater promptitude over the recovering area of the hand than over the intermediate zone between the touch and prick borders. Thus, although the quality of the sensation that can be evoked from those parts of the hand where sensibility to prick has returned, closely resembles that of the intermediate zone, the intensity, and, therefore, the extent of the innervation, has considerably increased.

At the close of this stage of recovery, all analgesia has disappeared, leaving the affected part of the hand in a condition of sensibility, with the following characteristics. Light touch cannot be appreciated. Two compass points, even widely separated, cannot be discriminated. Sensation is lost to temperatures between about 25° C. and 40° C.

Prick causes a widely diffused and disagreeable
sensation, and temperatures below 2 rmly produce
a sensation of ice cold, irrespective egree of cold
registered by the thermometer. A st tween 45° C.
and 50° C. will, when recovery is well be perceived
in most cases as warmth, but above vill be called
"hot," even in the earlier stages o , on account
of the "stinging" it produces, whe temperature
sensation be present or not.

In this condition, the affected e hand may
remain for several months. Then, i e has healed
well, the border for loss of light to nd to be no
longer so definite as before. At firs rtion nearest
to the wrist loses its sharpness and ss; then the
boundary on the palm or on the dorsu and becomes
indefinite. Gradually the whole pal the case of
the ulnar nerve, the back of the han sensitive to
light touch, the fingers alone rema sthetic. At
last, even the fingers regain their ser light touch.
One of the earliest signs of return of of sensation
is the power of discriminating int degrees of
temperature.

As soon as light touch begins to b ted over the
affected parts, water from about 35 ° C. again
produces a sensation of warmth, and emperatures
between 25° C. and 40° C. can be d ed, the one
being said to be warmer or cooler thar .

Month by month, the sensation ca inful stimuli
grows less and less diffused and loses g character.
Month by month, the power of distin etween two
compass points improves. But, if th s been com-
pletely severed, the sensibility of the es not as a
rule become normal for more than s. The old
border for loss to touch can still be m by a change
in the character of the sensation pro cotton-wool,
a change consisting in diminution o and wide
diffusion from the point stimulated. A ter method
of marking out this border is to drag a y across the
palm from normal to affected parts. inal line of

loss of touch the point becomes more painful, and the pain produced is widely diffused. This change in quality remains many months after sensibility to light touch has been restored to all the affected parts of the hand.

Under favourable conditions even these differences may disappear, and the sensibility of the affected area may become indistinguishable from that of the normal skin.

Should the wound have suppurated, or the nerve have been left unsutured, sensation may still return by the same stages, but the time of restitution will be prolonged. Whenever healing of the nerve is rendered less easy by want of apposition of the divided ends, or by unfavourable conditions in the wound, such as suppuration, the final recovery of sensation may be incomplete.

But the power of recovery possessed by a sensory nerve, even under the most unfavourable conditions, is remarkable.

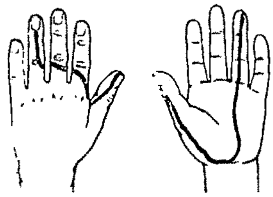


FIG. 5.

To show the area insensitive to cotton-wool and to intermediate degrees of heat and cold in Case 1. The whole hand was sensitive to prick and to the more extreme degrees of temperature.

We collected the names of all those who had been admitted to the London Hospital for injury to some peripheral nerve between the years 1892 and 1902. Several of these persons could not be traced, but many presented themselves, and were examined by us. Among them were fourteen cases where there was reason to suppose that the ulnar nerve had been divided, and eight of division of the median nerve. Out of these twenty-two patients, fourteen had recovered sensation so completely, that no difference could be discovered between the two hands. In one man, where primary suture had been performed four years before he came under our observation, no recovery had apparently taken place. He

was watched for a time, and, as no ent occurred,
further operation was suggested; f time he dis-
appeared. But, although our observ here incom-
plete, we can at any rate state with that in rare
cases no material recovery may take in four years.

Recovery may be arrested at the e first stage,
leaving the hand sensitive to pain an nore extreme
degrees of heat and cold, but inse light touch.
Such cases must be uncommon; we only one.

Case 1.—*Complete absence of sens ht touch and*
minor degrees of temperature, more than after primary
suture of the median nerve, in spite of res ility to prick.

On December 31, 1898, Henry S. wrist with a
broken bottle, and came to the London at once. The
tendons of the flexor sublimis, flexor car and palmaris
longus, had been divided, and the medi s completely
severed. The tendons and the nerve ited, and the
wound is said to have healed by first inte

When we first saw him in Februar said that his
hand only troubled him in cold weath abductor and
opponens pollicis acted voluntarily, and r n interrupted
current. Cotton-wool was not appreciat area shown
in fig. 5, and within these limits he was in- temperatures
between 20° C. and 40° C. Even 45° C ly said to be
anything but a touch. Yet ice and wat were every-
where called cold and hot correctly. N r the median
half of the palm could the two points of asses be dis-
criminated, even when they were separate nce of 2 cm.,
although, on the normal hand, he made s when they
were 1 cm. apart.

The whole of this area was sensitive t of a needle,
and the sensation so caused was not only greeable than
over the normal parts, but was widely d nning about
the hand." The borders of that part of t pplied by the
median nerve could be marked out, by no nts at which
a needle dragged across the palm bega this curious
diffused sensation.

If the hand becomes sensitive to p touch will
ultimately be appreciated over the affe ;[1] but this

---

[1] To this rule we have seen one exception only ted above).

restoration is not uncommonly incomplete. Any stimulus then causes a sensation which differs from that produced over normal parts in its diffuseness. Should the stimulus be of such a nature that it evokes pain, this pain will become a more prominent feature, and when a pin is dragged lightly across the skin, the patient withdraws his hand as soon as the border of the affected area is crossed, saying that it is more painful than over normal parts. Out of twenty-three cases, seven still showed this line of changed sensibility five years after the injury, and it was well marked in an old man whose ulnar nerve had been divided fifty-nine years before he came under our notice.

Whenever this line of change is present, the balance has not been re-established between that form of sensation evoked by a prick and that sensibility which responds to light touch. Light touch and the intermediate degrees of temperature can be appreciated. But, as the sensitiveness to these stimuli is less than normal, the diffuse and disagreeable characteristics of the earlier form of sensibility still intrude themselves, even with stimuli that are not painful over the normal skin. Whenever this line of change is well marked, the power of discriminating two compass points will be found to be diminished, a sign that the highest forms of sensation have not yet completely returned.

Case 2.—To show how the character of the sensibility of affected parts may remain changed six years after division of the median nerve, in spite of return of sensation to light touch.

In 1897, G. R. P. cut his left wrist with broken glass, and the wound was stitched at once without an anæsthetic. Sensation was completely lost, according to his account, over the index and middle fingers, and was changed in the palm. When we saw him in February, 1903, he complained that "although I can feel, I cannot define what I touch." He was a medical student, and daily noticed this inability of the left hand. He could not use his left hand for palpation, and if he was told to feel resistance in the abdomen he could not localise it, although he could appreciate the pressure against his hand. He complained that, in cold weather, the index and middle middle fingers became almost powerless.

The outer thenar group of muscles and opponens
pollicis) were wasted, but both muscl oluntarily and
reacted to the interrupted current.

The whole hand was sensitive to ligh h cotton-wool,
to the prick of a pin, and to temperat ° C. and 38° C.
But, if a pin was dragged across the palı ulnar towards
the radial aspect, the sensation chang ıdly at a line
corresponding to the border of the me On the radial
side of this line, the point caused a sensa spread widely,
and produced tingling in the fingers. stimulus was
repeated, he had an irresistible desire to part affected.

Over this area, on the radial half of he made two
mistakes in ten stimulations with the c ints at 2 cm.
At 1·5 cm., applied transversely, th were more
numerous $\left(\frac{1\,|\,6\ \text{R.}\ 4\ \text{W}}{2\,|\,7\ \text{R.}\ 3\ \text{W}}\right)$. But, on a simila e sound hand,
his answers were perfect when the com s were separ-
ated for not more than 0·75 cm.

In order that this line of change its appear-
ance in its characteristic form, the pa d must have
been sensitive for a considerable per recovery to
pain and to the more extreme forms ature alone.
Then, the old boundary for loss of light ll be marked
by a change in the character of the ı, for many
months after the whole hand has be sitive to all
forms of cutaneous stimulation. Bu er the two
forms of sensation have been restore *su*, this line
of change cannot be discovered.

No material part in this return of s light touch
can be attributed to overlapping fibre e uninjured
nerve trunk. For a comparison of tl of the area
insensitive to this stimulus produced ete division
of the ulnar or of the median nerve sh they must
overlap one another to a slight amou Out of the
nine cases where the ulnar nerve wa the anæs-
thesia occupied half the ring finger i ne-third in
three, and less than one-third in one more than
one-half the ring finger was insensitive ouch. Out
of twelve cases of division of the med , sensation
to light touch was lost over one-half t nger in six,
one-third in three, over less than a thi , and over

more than one-half in one instance. In no case, where either nerve was proved to have been divided, was the ring finger entirely unaffected. Thus, on the ring finger at any rate, the overlapping must be at most one-third of the breadth of the finger.

On the palm, the area insensitive to light touch has an outline which varies in each case. But here also the evidence points to no considerable overlapping between the supply of the median and ulnar nerves, as far as sensation to light touch is concerned.

The remarkable length of time required for the return of this form of sensation after complete division of the nerve, and the extraordinary fixity of the boundaries of the anæsthesia, all show that ultimate recovery is due to return of conduction, rather than to substitution by the overlapping fibres of the uninjured nerve.

But the ill-defined borders and the comparatively small extent of the total analgesia, and the fact that a large part of the palm rarely becomes insensitive to prick from a lesion of one nerve only, all point to much overlapping of the fibres that conduct pain impressions. Such overlapping should lead to rapid restoration of sensibility to prick, and in some cases possibly forms a factor when sensation returns with unusual rapidity. Commonly, no wide loss to prick on the palm follows division of the median nerve, because the fibres which conduct this form of sensation are supplied from both nerves. But, supposing the nerve supply of the median palm came overwhelmingly from the median, division of this nerve would produce at first total analgesia. This might rapidly pass away, to some extent, as soon as the few fibres of the ulnar nerve to the median palm became capable of supplying sufficient sensibility for the transmission of impulses. This certainly forms an important feature in the recovery of sensation to prick after division of the volar branch of the ulnar nerve.

Thus a girl of 17 (Case 24)[1] divided her ulnar nerve below the dorsal branch. The divided tendons and the ends of the nerve were dealt with the same day, and the wound healed

---

[1] Reported in full in the Appendix, p. 313.

by first intention. At first, sensatic was lost over
a small area in the centre of the n. This loss
rapidly disappeared, leaving an area hesia to light
touch over the palmar aspect of one lf fingers and
over the ulnar half of the palm. he remainder
of the summer, the condition of t improved, but
remained stationary throughout tl part of the
winter. Then, sensation to prick o deteriorate,
and the state of the nerve was tl xplored. At
the operation an extraordinary co s discovered,
which prevented all possibility of un upper end of
the nerve had been sutured to the endon of the
flexor carpi ulnaris, the lower end rve to one of
the tendons of the flexor sublimis di Thus, all the
return of sensation to prick, whic d during the
summer months, must have been du nnervation of
the parts affected by the dorsal bra ulnar nerve,
which had remained intact. The su deterioration
was probably caused by the numbi of the cold
weather on a part sensitive only to to extremes
of heat and cold.

With so much overlapping of n ly, complete
recovery of sensibility to prick migh ithout union
of the divided nerve, by a further devc f those fibres
in the uninjured nerve which norma the affected
parts. In areas where sensation to nly partially
lost, such substitution undoubtedly occ have shown
by the above example. But there is nce to show,
that restoration of sensation can be in analgesic
parts without union of the divided ne one instance
(Case 83), where the ulnar nerve ha erated upon
repeatedly and portions removed so as y to prevent
all chance of its reunion, sensibility showed no
signs of return. The fibres of the em to have
made no attempt to encroach on the a al analgesia,
produced by the original destruction o nerve.

Sometimes, it is necessary to div jured nerve,
after sensibility to prick has already b turn to the
hand, that more perfect union may be Wherever

such an operation has been performed, the parts that had begun to recover sensibility became again insensitive to prick, a proof that the recovery must have been due to union, however imperfect, of the divided nerve. Impulses from the recovering parts had passed up the injured trunk, and not up one of the normal nerves. This contention is supported by the following instance (Case 11).[1]

In May, 1901, a stonemason cut his right forearm with broken glass, dividing the median nerve. So little improvement had taken place up to April, 1902, that Mr. Eve determined to explore the wound. The two ends of the nerve were found to be widely retracted, and between them lay what appeared to be a strand of connective tissue. The ends were freshened and united. After the operation the extent of the total loss of sensibility to prick had distinctly increased, showing that the slight recovery which had taken place must have been due to the strand of tissue that was found between the two ends of the divided nerve.

§ 2.—*Recovery after Division of Particular Nerves.*

When all the cases of nerve injury to the hand are massed together, certain general principles emerge clearly. But as soon as each constituent group is analysed, the number of cases becomes so small that general conclusions are overwhelmed in the special conditions surrounding each particular instance. Nevertheless, we have arranged our records in tabular form and shall now consider more in detail the manner in which sensation is restored after injury to each of the nerves supplying the hand. On these tables, the time of recovery of each of the great forms of sensation[2] is expressed in days. But it must be remembered that in many cases these dates are necessarily only approximate. A man, whose median nerve had been united, would be asked to come on a particular day. But perhaps he was at work and refused, or had changed his address and did not receive

---

[1] Reported in full in the Appendix, vide p. 304.

[2] For the significance of the terms "Protopathic" and "Epicritic" see Chap. 16.

our request, or simply did not troul to obey, say-
ing his hand was "all right." Be uld be found,
or other arrangements made, sever might elapse.
When ultimately he presented hims mination, the
particular form of sensibility, which early returned
on his previous visit, would proba been restored
completely. What date are we to this return?
If we enter the return as complete c vious visit, we
shall have antedated it by perhaps ; if we say it
had returned by the date of his next s certain that
we shall overstate the time necess e restoration
of this form of sensation. Thus, alt the dates are
given in terms of days it must no ght that our
observations warrant any such prec ys have been
adopted as our unit solely to avoi kwardness of
fractions of a week.

(A) *Median Nerve.* [T

In six cases of primary suture, w vound healed
without complication, the period bet peration and
the first return of sensation averaged

Out of these six cases the shorte occupied was
44 days, the longest 92. It may justl ted, that sen-
sibility to prick might return over lar qually at the
same time, and that the date of an ob nution in the
size of the analgesic area does not the time at
which recovery begins. This is prob The loss to
prick has indefinite borders; it me an area of
varying extent, within which sensibil stimulus is
greatly lowered. For a time, consid overy might,
and almost certainly does, take pla t materially
pushing back the border of absol f sensation,
return of function being confined m the outlying
zone of diminished sensibility. Whe ian nerve is
divided, this is peculiarly liable to occ e sensibility
of the median palm to prick is always lowered by
such an injury, and it will depend upon of this loss,
whether the analgesia appears to be c the fingers

TABLE

Case	Date of Injury	Nature of Injury	Operation and Result
No. 3, W. B. ..	Dec. 19, 1901	Glass cut of wrist. Many tendons divided. Nerve seen to be divided (H. H. and J. S.)	Primary suture, Dec. Healed well
No. 4, A. C. ..	Dec. 22, 1902	Glass cut of wrist. No tendons divided. Nerve seen to be divided (J. S.)	Primary suture, Dec. First intention
No. 5, T. P. ..	Oct. 2. 1902	Knife cut of wrist. No tendons divided. Nerve seen to be divided below a high branch to the thumb muscles (J. S.)	Primary suture, Oct First intention
No. 6, S. H. .. [*Vide* Appendix, p. 300.]	Oct. 5, 1903	Razor cut of wrist. Many tendons divided. Nerve completely divided	Primary suture, Oc Healed well
No. 7, W. J. K.	Sept. 11, 1902	Glass cut of wrist. Many tendons divided. Nerve completely divided (J. S.)	Tendons sutured S 11. Nerve sut Sept. 19. Slight puration. In 1903, a stitch ab was opened and wound healed fir
No. 8, E. E. P.	Feb. 7, 1903	Cut with coal. No tendons divided. Nerve completely divided (J. S.)	Primary suture, Fe 1903. Wound h by granulation
No. 9, M. A. G.	Aug. 24, 1902	Glass cut of wrist. No tendons divided. Nerve completely divided (J. S.)	Primary suture, Au 1902. Wound h by granulation
No. 10, C. W. ..	Jan. 21, 1905	Glass cut of wrist. Palmaris longus tendon divided. Median nerve divided	Primary suture, Ja 1905. First inten
No. 11, D. J. T. [*Vide* Appendix, p. 304.]	May 6, 1901	Glass cut of forearm 3 cm. above fold of wrist. No tendons divided. At operation for secondary suture ends of nerve were found 4 cm. apart (H. H.)	Secondary suture, 16, 1902 (375 d First intention
No. 12, C. F. ..	Sept. 23, 1903	Cut with chisel 6 cm. above fold of wrist. Median nerve only divided (J. S.)	Secondary suture, 22, 1904 (153 day
No. 13, P. D. ..	.. .. ..	Glass cut of wrist. No tendons divided. At secondary suture ends of median nerve were embedded in fibrous tissue. No union (J. S.)	Secondary suture, 6, 1905

EDIAN NERVE.

Return of Sensation				
Protopathic	Epicritic	I		Muscles
egan March 7, 1902 (78 days). omplete June 4, 1902 (166 days)	Began June 11, 1902 (173 days). Complete Dec. 17, 1902 (363 days)	Line of c Feb. 1 Compassc at 1·5 c (530 da	ared ays). rfect 1903	Reacted to induced current July 2, 1902 (195 days). Voluntary movement Aug. 13, 1902 (237 days).
egan Feb. 4, 1903 (44 days). omplete July 12, 1903 (202 days)	Began Sept. 6, 1903 (258 days). Complete Nov. 18, 1903 (331 days)	Slight lin Compas- 1 cm. A days)	nly. at (972	Voluntary movement and reaction to induced current present Sept. 30, 1903 (282 days).
egan Nov. 26, 1902 (55 days). omplete May 24, 1903 (233 days)	Began Aug. 23, 1903 (324 days). Complete Jan. 13, 1904 (468 days)	Line of cl Feb. 26 Compasse fect at 1905 (94	red ys). per- 6,	No loss of power or reaction at any time.
gan March 30, 1904 (178 days). mplete April 27, 1904 (205 days)	Began July 8, 1905 (642 days)	.. ..	..	When first seen by us, Jan. 13, 1904, muscles acted slightly and reacted to induced current. Gradual improvement took place.
gan Nov. 26, 1902 (68 days). Remained almost stationary from Dec., 1902, to May, 1903. mpletely returned July 15, 1903 (299 lays)	Began July 15, 1903 (299 days). Terminal phalanges still affected, Aug. 19, 1903 (334 days)	Disappear tion Au line of and som sensibili phalange	va- ith ck, tic nal	Acted voluntarily and reacted to induced current July 15, 1903 (299 days).
gan May 13, 1903 92 days). mplete on Jan. 20, 904, when he was again found	Was lost again. On Aug. 24, 1904, he showed only a line of change (560 days)	.. ..	..	On Aug. 23, 1905 (924 days). Abductor and opponens pollicis did not act voluntarily or react to induced current.
gan Oct. 29, 1902 65 days)	.. ..	Disappeared tion.	a-	
gan March 15, 1905 54 days). nplete June 29, 1905 158 days).				
gan Dec. 21, 1902 220 days). nplete Jan. 25, 1903 254 days)	Began Jan. 31, 1904 (625 days). Complete April 10, 1904 (695 days)	Line of cha With com everythin (1,099 da	t. n. ”	Slight voluntary action and feeble reaction to induced current May 24, 1903 (373 days).
gan April 5, 1904 12 days). nplete Aug. 28, 1904 87 days)	Began June 18, 1905 (469 days)	.. ..	..	On Feb. 26, 1905 (369 days), muscles reacted to interrupted current. On June 18, 1905 (481 days), they reacted voluntarily.
an March 8, 1905 30 days). nplete Aug. 27, 1905 202 days).				

or to occupy the greater part of the palm supplied by the median nerve.

If the nerve has been reunited and the wound has healed well, the area of total loss to prick will begin to grow smaller in about 7 to 11 weeks, and the palm will then recover rapidly. Three to eight weeks later, total analgesia will be found on the fingers only; but here the skin may remain insensitive, especially over the terminal phalanges, for a considerable period. Finally, the whole of the affected area becomes sensitive to prick in about seven months, or 200 days, the average of four uncomplicated cases, in which the records are perfect, was 190 days.

We have already pointed out that the area of absolute analgesia is in some cases confined to the fingers; but, in many instances, it occupies a greater or less extent of the radial half of the palm of the hand. We believe that when absolute loss of sensation to prick is found on the palm, recovery begins at a somewhat earlier date than if this loss is confined to the fingers.

Any want of health in the wound retards recovery to a remarkable degree. In Case 7, slight suppuration took place in the original wound, which appeared ultimately to heal. Sensation began to return after 68 days; then no further recovery took place for five months. During this time, a swelling appeared on the site of the injury which was found to contain pus, evidently due to contamination of one of the deep stitches. In consequence of this deep suppuration, the return of sensation to prick was prolonged over a period of 230 days, rapid recovery following the evacuation of a small abscess.

Light touch began to be appreciated at times which varied from 173 to 324 days after suture of the nerve. Among four complete cases, the average time was 262 days. Here, again, the figures are only approximate; for, during the early stages of recovery, the condition of sensibility to light touch depends on circumstances out of the control of the observer. If the day is bright and warm and the patient in good bodily condition, cotton-wool may be appreciated faintly, but with certainty, on the palm of the

hand. A week later, in the bitter rly spring, the
same parts may be entirely insensit t touch. The
length of the time required before li (cotton-wool)
could be appreciated on every pa affected hand
varied from 331 to 468 days after n average in
three cases of 387 days.

But this in no way completes th of sensation.
Long after light touch can be appre r the affected
area, all stimuli cause a more disa ensation than
over normal parts. When a pin is lightly across
the skin, the borders of the area th ce anæsthetic
can still be recognised by the char racter of the
sensation produced by the point. A a successful
case, even this line of change disap e have been
able to follow two cases only up to th on; in No. 3
the line of change disappeared fron ed hand 419
days after suture of the nerve. This usually early,
for in No. 5, the line of change was shed for 877
days after the injury, and in two pa om we have
watched for over two years, this sign ive sensation
is still present.

Whenever this line of change is he apprecia-
tion of two compass points is defecti patient only
recovered complete power of discrim etween two
points, whilst in one it still remains d 45 days after
suture when the points are not more apart.

Neither simultaneous injury to te paralysis of
the muscles supplied by the median any obvious
effect upon the rapidity of the return o n. The most
satisfactory and most uniform return e in Case 3,
where the tendons were injured and ralysed. In
another instance (Case 5), the muscu was spared
and no tendons were divided; but re upied rather
more than the average time. On Tabl ee instances
of secondary suture of the median ne recovery of
sensation ran a course so different in cases, that
the consideration of the effect of sec ure will be
postponed, until we have analysed th of sensation
after injury to the ulnar nerve.

(*I*) *Ulnar Nerve.* [*Table II.*]

We have been able to watch the recovery of sensation in four cases where the ulnar nerve was divided at the wrist. In three, the wound healed without complication, and sensation first began to return on an average in 109 days (92 days, 104 days, 131 days). The whole of the affected parts had become sensitive to prick in from 166 to 181 days—an average of 171 days.

TABLE I

(*A*) TOTAL NERVE TRUNK.

Case	Date of Injury	Nature of Injury	Operation and Resu
No. 14, J. S. ..	June 26, 1903	Glass cut 6 cm. above fold of wrist. Tendon of flexor carpi ulnaris and innermost tendon of flexor sublimis divided. Nerve completely divided (J. S.)	Primary suture, 26, 1903. Heale healthy granulati
No. 15, E. R. ..	July 29, 1903	Glass cut 2·5 cm. above fold of wrist. Tendons of flexor carpi ulnaris and ulnar tendons of flexor sublimis divided. Nerve completely divided (J. S.)	Primary suture, Jul 1903. Conside suppuration. He by granulation
No. 16, A. P. ..	June 11, 1903	Glass cut of wrist. All tendons divided except flexor carpi radialis and radial tendons of flexor sublimis. Nerve completely divided (J. S.)	Primary suture, 11, 1903. Wo healed by first i tion
No. 17, J. M. ..	Aug. 26, 1904	Glass cut of wrist. Flexor carpi ulnaris and ulnar nerve divided	Tendon and nerve tured, Aug. 26, 19
No. 18, W. W...	July 16, 1902	Glass cut of elbow. Extensor communis digitorum wounded. Ulnar nerve divided	Nerve sutured, Jul 1902. Healed by intention
No. 19, L. C. .. [*Vide* Appendix, p. 309.]	. .	Injury to elbow-joint in childhood. Bony outgrowths pressed upon nerve during his work; formation of fibroma of nerve	June 17, 1904. Rese of 4·5 cm. of ner elbow and sutu divided ends (J.

Unfortunately, we are able to L[illegible] ward two cases only to determine the date at which se[illegible]y to light touch returns after uncomplicated primary suture of the whole ulnar nerve. In one of these the period was 166 days, in the other 172 days—an average of 169 days or 24 weeks.

In one instance only were we able to follow the patient to complete recovery. Here the affected parts of the hand became sensitive to light touch in 278 days and the "line of change" had disappeared in 590 days; at this date he gave

LNAR NERVE.

Return of Sensation			Muscles
Protopathic	Epicritic	Final Result	
gan Nov. 4, 1903 (131 days). mplete Dec. 9, 1903 167 days)	Began Dec. 16, 1903 (172 days). Complete March 30, 1904 (278 days)	Line of change gone Feb. 5, 1905. Compasses perfect at 1 cm. (590 days)	Reacted to induced current Oct. 30, 1904 (492 days). Feb. 5, 1905, muscles acting perfectly (590 days).
gan Dec. 2, 1903 (127 days). mplete June 12, 1904 320 days)	Began June 12, 1904 (320 days). Complete Aug. 27, 1905 (760 days)	Line of change present Aug. 27, 1905	June 12, 1904 (320 days), 1st dorsal interosseous and adductor pollicis acted voluntarily. First dorsal interosseous reacted to strong induced current.
gan Sept. 23, 1903 104 days). arly complete when ie disappeared Dec.), 1903 (131 days)	Recovery had not begun Dec. 9, 1903, but was complete when he was next seen, June 29, 1904 (383 days)	On August [illegible], 1904, line of change still present, and the compasses were badly appreciated at 2 cm. (418 days)	All muscles reacted to induced current June 29, 1904 (383 days)
gan Nov. 23, 1904 92 days). mplete Feb. 8, 1905 166 days)	Began Feb. 8, 1904 (166 days)	.. ..	No recovery of muscles.
gan Nov. 26, 1902 117 days). nplete Jan. 28, 1903 180 days)	Began May 13, 1903 (287 days). Complete Jan. 6, 1904 (525 days)	On June 18, 1905, line of change gone; compasses good at 2 cm., uniformly wrong at 1 cm. (1053 days)	July 3, 1904 (702 days), all interossei acted and reacted to the interrupted current. Muscles acted voluntarily.
gan Sept. 14, 1904 89 days). nplete Aug. 16, 1905 425 days)			

TABLE II.-

(B) DORSAL BRANCH INTACT.

Case	Date of Injury	Nature of Injury.	Operation and Result
No. 20, A. L. ..	Jan. 25 1903	Glass cut of wrist. Nerve divided ..	Primary suture, Ja 26, 1903. Healed b healthy granulation
No. 21, H. E. ..	July 11, 1901	Cut of wrist with a stoneware jar. Tendon of flexor carpi ulnaris divided. Nerve completely divided (J. S.)	Primary suture, July 1 1901. Healed by fi intention
No. 22, H. W. ..	July [illegible], 1904	Glass cut of wrist. No tendons. Nerve divided below dorsal branch	Primary suture, July 1904. Healed by gra ulation
No. 23, K. W. ..	Between Aug. 26-3[illegible], 1901	Glass cut of wrist. At operation for secondary suture nerve was found completely divided (H. H.)	Secondary suture, Sep 24, 1901 (about days). Healed by fi intention
No. 24, E. A. .. [*Vide* Appendix p. 313.]	March [illegible] 1902	Glass cut of wrist. At operation for secondary suture it was found that divided nerve had been stitched to tendons (J. S.)	Secondary suture, Ju 22, 1903 (502 day Healed by first inte tion

good answers to the compass test, even when the points were separated to 1 cm. One other patient, who had been watched for a time and then disappeared, was rediscovered 383 days after the injury; at this time he could appreciate light touch everywhere over the parts affected, but a marked line of change was present and the compass test was defective at 2 cm.

The ulnar nerve, in consequence of its exposed position at the elbow, is subject to injuries at a point at least 25 cm. distant from the wrist. This seems to make little material difference to the length of time required for the return of sensibility to prick. In No. 18, this form of sensation began

NAR NERVE.

Return of Sensation			Muscles
Protopathic	Epicritic	Final Result	
begun June 10, 03 (135 days), and as complete Aug. 5, 03 (192 days)	Began Sept. 16, 1903 (234 days). Complete March 23, 1904 (424 days)	On June 8, 1904, line of change still present. Compasses entirely wrong at 2 cm. (500 days)	Acted voluntarily, and reacted to induced current Dec. 9, 1903 (319 days).
n Aug. 13, 1901 3 days). plete Nov. 13, 1901 25 days)	Had not begun to return on Dec. 20, 1901	Died of malignant disease of the liver, Dec. 23, 1901	On Nov. 13, 1901, no muscles acted voluntarily or reacted to induced current.
already begun on ig. 10, 1904 (35 ys). plete Sept. 28, 1904 4 days)	Began Nov. 16, 1904 (133 days). Complete Feb. 8, 1905 (217 days)	June 21, 1905, compasses perfect at 1·5 cm.; badly appreciated at 1 cm.	Feb. 8, 1904 (217 days), 1st dorsal interosseous reacted to induced current, and showed the first traces of voluntary movement.
n seen by us there s no loss of this m of sensibility days)	Began Feb. 26, 1902 (154 days). Complete June 11, 1902 (258 days)	Dec. 7, 1904, line of change present. Compasses at 2 cm. everything called "two"	Nov. 7, 1902 (408 days), 1st and 2nd dorsal interossei acted voluntarily, and all ulnar muscles reacted to induced current.
n Dec. 2, 1903 3 days). plete Jan. 13, 1904 6 days)	Began March 30, 1904 (252 days). Complete Sept. 21, 1904 (426 days)	Aug. 16, 1905, line of change present. Compasses perfect at 2 cm.; at 1·5 cm. everything called "two" (755 days)	July 27, 1904 (370 days), 1st dorsal interosseous acted voluntarily. Adductor pollicis and abductor minimi digiti reacted to induced current.

to return in 117 days, and the whole affected parts had become sensitive in 180 days. In No. 19, where a portion of the nerve had been excised at the elbow and the ends reunited, sensation began to return in 89 days, and in 425 days had returned completely.

But injury at the elbow seems to cause a material delay in the final restitution of sensibility to light touch compared with the period required, when the nerve has been wounded at the wrist. In No. 18 light touch was first appreciated 287 days after the nerve had been sutured, and the whole area affected did not become sensitive to cotton-wool for 525 days. In this instance the "line of change" did not disappear for nearly three years (1,053 days).

When the dorsal branch remains intact, it is usually difficult to determine the date at which sensibility to prick begins to return. For the area of total loss is mostly so small, and is at the same time so variable according to the temperature of the hand and the general condition of the patient, that it is sometimes impossible to say, if the obvious increase in sensibility is due to recovery of the nerve or only to more favourable general conditions.

The date at which sensation to prick is completely restored can be determined more satisfactorily. The analgesia retreats to the terminal phalanx of the little finger, and

TABLE III.—DIVISION

Case	Date of Injury	Nature of Injury	Operation and Result
No. 25, A. H. ..	Dec. 24, 1902	Cut wrist with soda-water syphon. All structures divided down to the bone (J. S.)	Primary suture, De 24, 1902. Suppurat
No. 26, M. L. ..	Oct. 26, 1902	Cut wrist with jug. All structures divided down to the bone	Primary suture, Oct. 1902. Suppurated
No. 27, A. W. ..	Mar. 10, 1901	Glass cut of wrist. Both nerves divided	Primary suture at other hospital. S purated
No. 28, G. B. .. [*Vide* Appendix, p. 321]	Sept. 24, 1902	Glass cut of wrist. Tendons were sutured, but not nerves	Secondary suture, A 17, 1903 (205 da Both nerves found vided. First intent

it is easy to discover if this part is sensitive or not. The average period necessary for complete recovery of sensation to prick was 133 days, but the individual variation is great, extending in our cases from 84 days[1] to 192 days.

Division of the ulnar nerve below its dorsal branch causes loss of sensation to light touch over a considerable area of the palm. In two instances of primary suture of the nerve, this form of sensibility began to return in 234 and 133 days (an average of 183 days). The whole affected hand had become sensitive to light touch 424 and 217 days after suture.

DIAN AND ULNAR NERVES.

Return of Sensation			Muscles
Protopathic	Epicritic	Final Result	
l begun Mar. 8,)03 (74 days). ıplete May 13, 1903 10 days)	Began July 12, 1903 (200 days). Complete Sept. 23, 1903 (273 days)	Changed sensation had not entirely disappeared July 8, 1905 (726 days). Compasses perfect at 2 cm.	Sept. 23, 1903 (273 days), interossei and abductor pollicis acted voluntarily and 1st dorsal interosseous and abductor minimi digiti reacted to the interrupted current.
an Mar. 5, 1903 28 days). ıplete Aug. 19, 1903)5 days)	Began Aug. 19, 1903 (295 days). Complete Dec. 2, 1903 (399 days)	Line of change disappeared Feb. 17, 1904 (475 days). Aug. 23, 1905, compasses still bad at 2 cm.	Sept. 2, 1903 (308 days), opponens and abductor pollicis reacted to strong interrupted current. Feb. 17, 1904 (475 days), first dorsal interosseous reacted to interrupted current. July 13, 1904 (621 days), all muscles reacted to interrupted current. Oct. 20, 1903 (356 days), opponens and abductor acted voluntarily.
. 10, 1901 (245 ys), when first ən by us. Protothic sensibility d returned	Began Jan. 24, 1902 (320 days). Complete Mar. 8, 1903 (728 days)	Line of change gone May 24, 1903 (804 days). With compasses at 3 cm. everything called "two," Aug. 21, 1905	Nov. 5, 1902 (605 days), opponens and adductor pollicis acting voluntarily; did not react to induced current. Mar. 8, 1903 (728 days), all muscles acted voluntarily and reacted to induced current.
n Sept. 27, 1903 3 days). plete Dec. 20, 1903 7 days).	Began April 10, 1904 (358 days).		

---

[1] In a boy of 10 years of age.

(C) *Median and Ulnar.*

In spite of the severity of the lesion and the great extent of the analgesia it produces, sensibility to prick began to be restored to the affected palm in two instances 74 and 128 days after primary suture, and the whole hand became sensitive to prick in 140 and 295 days. One patient (No. 27) was seen by us for the first time 245 days after primary suture, and by this time all analgesia had disappeared.

Thus, although the material at our disposal is small, it is evident that the length of time necessary for the restoration of sensibility to prick after division of both nerves does not materially exceed that required after division of the ulnar only. This result is particularly noteworthy, as in all the patients with division of both nerves, from whom these numbers were drawn, the wound suppurated.

As far as the return of sensibility to prick is concerned, it seems to matter little whether one or both nerves have been divided. But simultaneous division of the two nerves materially delays the return of sensation to light touch. On an average, in three instances, this form of sensibility did not begin to return until 271 days, and was not universally restored to the affected parts until 470 days, after suture. However, it must be remembered that in all these patients, the wound suppurated. But in spite of this objection, we are inclined to believe that simultaneous division of both nerves materially retards the return of sensibility to light touch.

(D) *Summary.*

Our records are sadly incomplete, and it is not possible to determine, with the accuracy we could wish, the period requisite for the various stages of recovery. But it will be well, as far as possible, to summarise our results with the warning that, since satisfactory cases are so few, the interjection of some unrecognised circumstance, even in a single instance, may have materially upset any of the following averages :—

	Protopathic		Epicritic	
	Began	Complete	Began	Complete
Ulnar with dorsal branch intact	?	133	183	320
Complete ulnar nerve	109	171	169	278
Median nerve	65	190	262	387
Median and ulnar nerves ..	101	217	271	470

From this table it will be seen that the earliest recovery of sensibility to prick occurs after primary suture of the median nerve. For reasons given above, we have purposely neglected the date at which the restoration of sensation began in those cases where the ulnar nerve was divided but the dorsal branch remained intact. It is after such an injury that the analgesia disappears completely with the greatest rapidity. Even when both nerves had been divided, sensibility to prick was restored in 217 days, or a little over six months.

The length of time necessary for complete recovery of sensibility to cotton-wool and to the intermediate degrees of temperature varied somewhat, but occupied about a year in most cases. Any defect in healing, particularly if it leads to the formation of pus, or of fibrous tissue around the nerve, materially hinders the return of the higher forms of sensation.

CHAPTER 3.—RECOVERY OF SENSATION AFTER INCOMPLETE DIVISION OF THE NERVES OF THE HAND.

If a nerve has been bruised, or incompletely divided, it may become entirely incapable of conducting impulses, and the loss of sensation at first may resemble that which follows complete division. But, when the continuity of the nerve has not been destroyed, recovery may take place, not

TABLE IV.—MEDIA

INCOMPLETE DIVISION OF THE MEDIAN NERVE.

Case	Date of Injury	Nature of Injury	Operation and Result
No. 29, C. B. .. [*Vide* Appendix, p. 302.]	Sept. 20, 1902	Cut wrist with glass bottle. Median nerve found swollen and redder than normal; on ulnar side it had been cut into. Tendon of flexor sublimis to index had been divided (J. S.)	Wound explored Sept. Nerve left untouch Tendon sutured. F intention
No. 30, D. B. ..	July 31, 1904	Glass cut of wrist. Median nerve incompletely divided. Four tendons of flexor sublimis divided	Tendons and nerve tured Aug. 1, 19 Healed by heal granulation
No. 31, H. B. ..	Dec. 18, 1904	Glass cut of wrist. Median nerve incompletely divided	On Dec. 18, 1904, wo stitched without anæsthetic. On 15, 1905, explored nerve examined (J.
No. 32, A. S. ..	April 22, 1905	Glass cut of wrist. Median nerve cut into on ulnar side. Some tendons of flexor sublimis divided	Wound in nerve sut and tendons reun April 22, 1905. intention

INJURY TO MEDIAN NERVE.

Case	Date of Injury	Nature of Injury	Result of Injury
No. 33, H. E. T.	Dec. 26, 1903	Fractured forearm. Splint pressure. Volkmann's contracture	Complete loss of fur in median nerve

ERVE.

RETURN OF SENSATION			Muscles
Protopathic	Epicritic	Final Result	
gan Oct. 22, 1902 32 days). mplete July 12, 1903 294 days)	Began Oct. 22, 1902 (32 days). Nearly complete July 12, 1903 (294 days)	Patient could not be found after July 12, 1903	Did not cease to act voluntarily. Did not react to interrupted current between Oct. 22, 1902, and Feb. 11, 1903 (143 days). To galvanism reactions were practically normal throughout.
gan Sept. 7, 1904 37 days). nplete Dec. 14, 1904 35 days)	Began Sept. 7, 1904 (37 days). Complete Dec. 14, 1904 (135 days)	Line of change well marked Aug. 16, 1905. Compasses good at 1·5 cm. (380 days)	Sept. 7, 1904 (37 days), abductor and opponens acted voluntarily, but did not react to the interrupted current until Oct 26, 1904 (85 days). To galvanism reactions were practically normal throughout.
an Mar. 1, 1905 3 days). ost completely re- rned, Aug. 16, 1905 68 days)	Began Mar. 22, 1905 (94 days). Almost completely returned Aug. 16, 1905 (168 days)	.. ..	Mar. 1, 1905, muscles acted voluntarily and reacted to interrupted current (73 days).
an May 31, 1905 8 days). plete Aug. 30, 1905 29 days)	Began Aug. 30, 1905 (129 days)	.. ..	May 31, 1905 (38 days), reacted to interrupted currents. July 14, 1905 (82 days), acted voluntarily.

Protopathic	Epicritic	Final Result	Muscles
n July 3, 1904 38 days). plete Feb. 26, 1905 5 days)	Began July 3, 1904 (188 days). Complete May 6, 1905 (493 days)	Compasses perfect at 1·5 cm. Aug. 27, 1905 (606 days).	July 24, 1904 (208 days), muscles reacted to interrupted current.

only more rapidly, but also in an entirely different manner from that observed after complete division. At the end of a period varying with the severity of the injury, sensation to prick begins to return. Approximately at the same time, light touch also begins to be appreciated over those parts of the area previously insensitive to this stimulus, which lie nearest to the wrist.

Thus, if a nerve is injured, but not divided, both sensibility to prick and to light touch begin to return together, in contradistinction to the order in which sensation is restored when the continuity of the nerve has been completely destroyed.

As recovery progresses, the two forms of sensibility continue to advance *pari passu*. For instance, if the median nerve has been injured, the gradual diminution of the analgesia on the fingers is accompanied by an equivalent increase in the sensibility of the palm of the hand to light touch.

With the power to appreciate light touch, the affected parts become sensitive to intermediate degrees of temperature. In fact, restoration of this form of sensibility over the proximal parts of the palm, at a time when the analgesia has scarcely begun to diminish in extent, is one of the earliest indications that the nerve has not been completely divided.

With the return of sensation to light touch and to the intermediate degrees of temperature, comes a coincident improvement in the answers given by the patient to the compass test. The power of discriminating two points is not only restored more rapidly, but more often returns completely than when the continuity of the nerve has been destroyed.

With regard to the principle underlying the manner in which sensation is restored, it matters little whether the nerve has been bruised or incised. We have, therefore, gathered together all the instances of injury and incomplete division on the same table.

But these partial injuries may produce very varying results. Sometimes every function of the nerve is destroyed for a time; sensation is lost and the muscles are paralysed, of several posterior roots abolishes sensation to prick over an area larger and more sharply defined than that which becomes insensitive to light touch. Moreover, this insensibility to prick is accompanied by an inability to appreciate temperatures below 15° C. and above 60° C., although 40° C. and 23° C. may appear definitely warm and cool.

CHAPTER 7.—NERVE SUPPLY OF THE LOWER LIMB.

§ 1.—*The Sole of the Foot.*

The nerves of the lower limb are much less frequently injured than those of the arm and hand, and most of the injuries fall either upon the sciatic or upon the external popliteal. Wounds, that divide the nerves to the sole of the foot in the neighbourhood of the ankle, are so uncommon that it is impossible to determine the distribution of the internal and external plantar; but the following instance shows in a remarkable way the nature and extent of the sensibility supplied by the posterior tibial nerve.

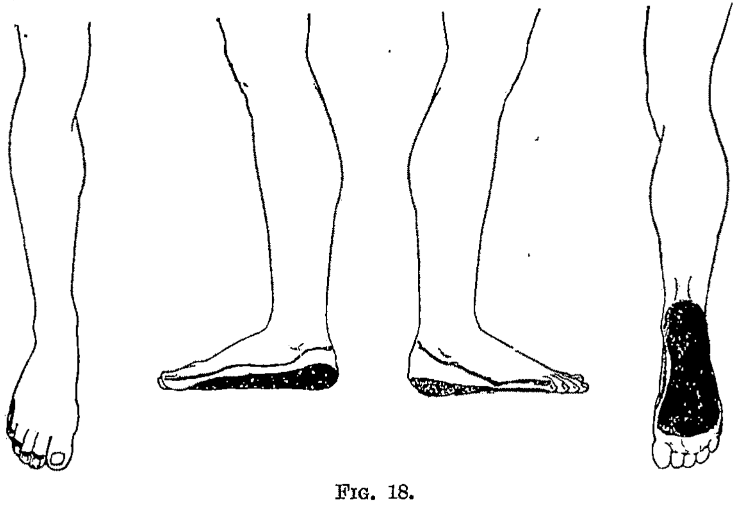


FIG. 18.

To show the area of cutaneous insensibility produced by division of the posterior tibial nerve (Case 54). Before the operation on August 15, 1902, the whole of the parts enclosed by the single line were insensitive to light touch, and to the minor degrees of heat and cold. After the operation the loss of sensibility to these stimuli remained unaltered, but the parts in black became insensitive to all cutaneous stimuli.

Case 54.—Division of the posterior tibial nerve at the ankle.

On May 16, 1901, J. T., while serving in South Africa, was shot through the right leg. The foot became "numb" and useless at once, but fourteen days later "feeling" came back. This return of sensation was associated with so much pain in the sole of the foot that the condition of the nerve was explored from the popliteal space, two months after the injury. He returned to England on August 22, and was sent to Devonport Hospital. There the posterior tibial nerve was divided just behind the internal malleolus, "in order to stop the swelling of the foot," and in consequence the whole sole became "numb."

In August, 1902, he came under the care of one of us at the London Hospital. The scar caused by the entry of the bullet lay just anterior to the tendon of the biceps femoris, 3 ins. (7·5 cm.) above its insertion; the wound of exit was 4 ins. (10 cm.) above the head of the tibia over the inner group of hamstring muscles. In the centre of the popliteal space was a well-healed surgical scar, a relic of the first operation, and behind the internal malleolus lay another scar, 1¼ ins. (3·5 cm.) in length, due to the operation in Devonport Hospital.

All the muscles below the knee reacted to the interrupted current, but the only movement of which they were capable was extension of the foot.

He complained that his foot was sore when he put it to the ground, and that this prevented him from walking.

The right sole was entirely insensitive to touch with cotton-wool, a stimulus which he easily appreciated over the whole of the normal foot. Sensibility to prick was nowhere lost, but, wherever light touch was defective, a prick caused increased discomfort, and was associated with a sensation of pins and needles. The whole of this area was insensitive to water at 15° C. and at 40° C., but water at 50° C. was said to be "very hot," and ice produced a sensation of numb, tingling cold. Over the sole of the right foot, the points of the compasses could not be discriminated when separated for 6 cm., whilst on the sound foot he made two mistakes only when they were 2 cm. apart.

We concluded from the physical signs that in this case the bullet had injured the sciatic nerve just above the popliteal space. Sensibility to pain was probably abolished for a few weeks only, and then, on the way to recovery, the foot became over-sensitive to all painful stimuli. Gradually the nerve regained its power of conduction, and the muscles their normal reaction. But meanwhile, in consequence of division of the posterior tibial nerve, the sole of the foot had become insensitive, and he was in a worse position than he would have been without the operation.

On August 15, 1902, Mr. Barnard cut down on the structures behind the internal malleolus. A mass of firm fibrous tissue was exposed, and when this was dissected away, the two ends of the nerve were seen, the upper a bulbous mass and the lower spread out over, and closely adherent to, the vein. The nerve was dissected with difficulty from the vein, and when completely free was found to consist of an upper and a lower end connected by dense fibrous tissue. This intervening tissue was incised at several points, until the normal fibres of the central end were exposed. The distal end of the nerve was treated in a similar way until nerve fibres in considerable number became visible. The two ends of the nerve were then united together.

After this operation all pain disappeared, the extent of the loss of sensation to light touch remained unaltered, but sensibility to prick was lost over a wide extent of the sole (fig. 18). Over the whole of this area all degrees of temperature were unappreciated. When passing from normal to affected parts of the foot, minor degrees of temperature, such as 20° C. and 40° C., were no longer appreciated as soon as the border for loss to light touch was passed.

This case shows that the posterior tibial nerve supplies the sole of the foot with all forms of cutaneous sensibility. On the inner side, the border at which sensibility ceases is a well-defined line identical for all forms of sensation. But over the outer side, both the area of loss of sensation to light touch and that of the analgesia merge gradually into parts of normal sensibility, the loss of light touch exceeding in extent that of the loss to prick. From their plantar aspect, the toes were insensitive to light touch, but remained sensitive to prick.

Here also, as in the hand, recovery of sensation after complete division of the nerve began with a return of sensibility to painful stimulation, and to ice, and water at 50° C. The parts affected remained insensitive to light touch and to minor degrees of heat and cold, and two points could not be differentiated, even when separated to a distance of 6 cm.

That this return of sensibility was not due to substitution from the surrounding nerves is shown by the complete loss of all sensation produced by again dividing the affected nerve in order that its two ends might be sutured together.

§ 2.—*Loss of Sensation produced by Injury to the Nerves of the Leg.*

Division of the external popliteal nerve, below the point at which its lateral cutaneous branch is given off, causes loss of sensibility to light touch over a considerable portion of the outer side of the leg and over the whole of the dorsum of the foot (fig. 18, A). Within these limits sensibility to prick is evidently diminished, but is nowhere lost entirely, except over the dorsum of the foot (fig. 18, B). These figures, illustrating the loss of sensation produced by division of the external popliteal, were obtained from the following case.

Case 55.—*Injury to the external popliteal; resection and suture of the nerve.*

Joseph B. was admitted to the Poplar Hospital, under the care of Mr. Rigby, in July, 1900, with a compound comminuted fracture of the left tibia and fibula. Whilst falling, the right leg turned under him and he sustained a slight wound over the external condyle of the right femur. This wound healed rapidly, but three weeks after admission the patient was found to show signs of paralysis of the right external popliteal nerve. In spite of massage, the leg was still paralysed when he first came under our notice in October, 1901. Mr. Rigby therefore explored the nerve at the site of the injury and found it hard and firm, embedded for a distance of 1½ ins. (4 cm.) in fibrous tissue; it had evidently been partially ruptured, and the upper end was bulbous and united to the lower portion by a strand of fibrous tissue. An inch and a half (3·75 cm.) was excised and the two freshened ends were reunited with silk sutures.

The paralysis of motion and sensation was in no way increased by this operation, proving that the pre-existing condition was due to complete functional division of the nerve.

The whole anterior tibial group of muscles (tibialis anticus, extensor longus digitorum and extensor hallucis), together with the peronei, were paralysed and had lost their reaction to the interrupted current. The flexor muscles of the toes and the muscles of the calf acted well.

The loss of sensation to light touch and to the painless interrupted current, shown on fig. 18, A, was bounded towards the shin and on the inner side of the dorsum of the foot by a definite line, but above, merged gradually into parts of normal sensibility. Sensation to prick was disturbed over an area of smaller extent (fig. 18, B), but the boundary on the inner side of the dorsum of the

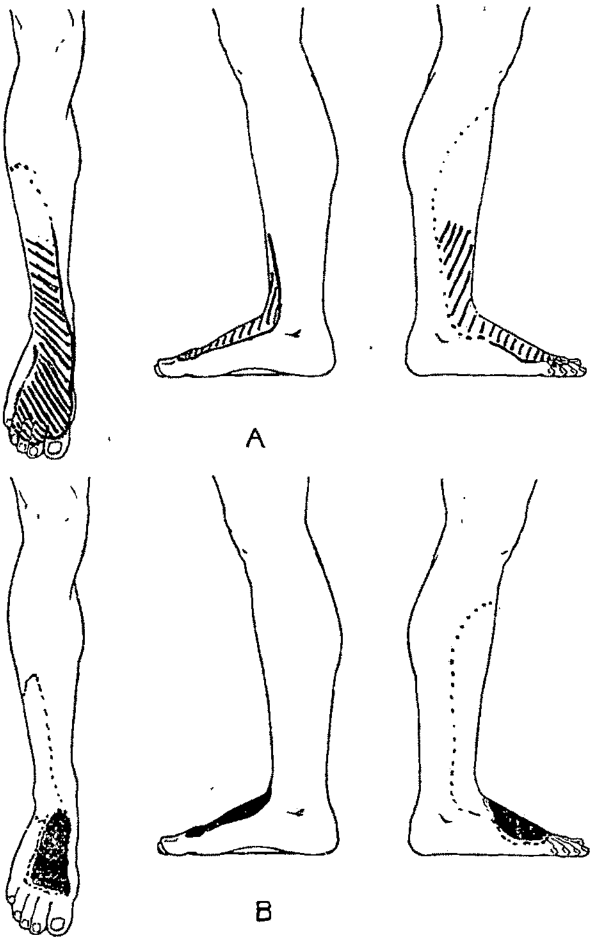


FIG. 19.

To illustrate Case 55.

A shows the extent of the leg insensitive to light touch, and the intermediate degrees of temperature after division of the external popliteal below its lateral cutaneous branch. The area of complete insensibility is shaded.

B shows the extent of loss of sensation to prick. The area of total analgesia is marked in black.

foot coincided with that of loss of light touch. Ice and water above 50° C. were not appreciated over the analgesic area, and he was unable to discriminate minor degrees of heat and cold over those parts where sensibility to light touch was destroyed. Deep touch and pressure were recognised everywhere over the affected parts. Over the dorsum of the sound foot, he could appreciate the two points of the compasses correctly when 4 cm. apart; at this distance he failed entirely over a similar part of the affected foot.

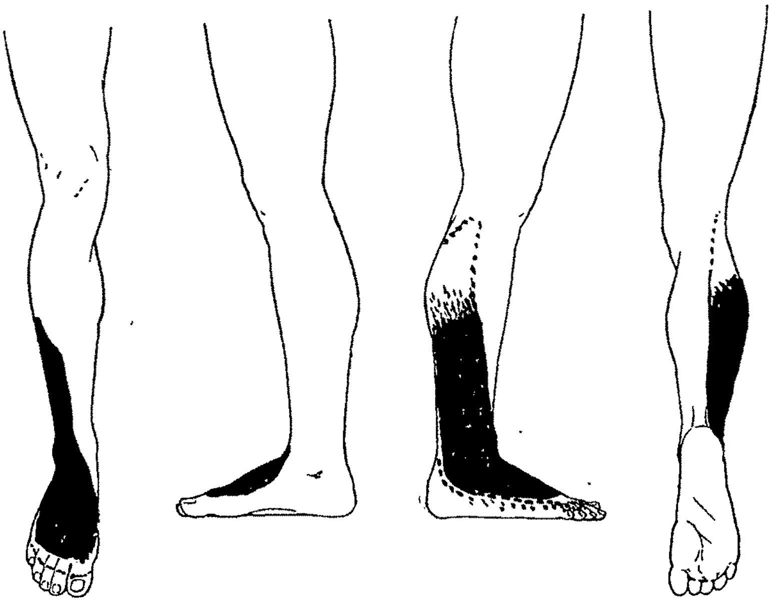


FIG. 30.

To illustrate the loss of sensation produced by division of the external popliteal above its lateral cutaneous branch (Case 56).

Total cutaneous insensibility is marked in black; this area merges above and below into parts sensitive to prick, but insensitive to light touch. The loss of sensation to light touch is enclosed in a dotted line. Above and on the outer side of the foot this area of loss to light touch merges into parts of normal sensibility.

When the external popliteal is divided below the origin of its lateral cutaneous branch, the posterior (sural) border of the loss of sensation is always ill defined in contrast to the astonishing definiteness of the anterior border. But when the continuity of the whole of this division of the sciatic is destroyed above the point where the lateral branch is given off, the posterior border in the calf becomes as definite as that on the shin (fig. 20).

Case 56.—Complete division of the external popliteal nerve above the origin of its lateral branch.

On December 25, 1901, whilst serving in South Africa, Charles G. was shot through the right thigh with an explosive bullet. He fell and at once discovered that he could not move his leg.

When we saw him at the Royal Victoria Hospital, Netley (March 26, 1902), the wound of entry on the posterior aspect of the thigh 5 ins. (12·5 cm.) above the centre of the popliteal space had healed. On the outer surface of the thigh was a triradiate scar nearly 4 ins. (10 cm.) from end to end, in the centre of which lay a small area not completely healed.

The anterior tibial and peroneal groups of muscles were paralysed, much wasted, and did not react to the interrupted current. The foot could be inverted and the toes flexed; the calf muscles, though somewhat wasted, contracted strongly and reacted to the interrupted current.

Sensibility to light touch was lost within the dotted line on fig. 20. It will be evident how closely its anterior and posterior borders correspond with the extent of the loss of sensation to prick marked in black. The upper border and the border on the outer side of the foot were indefinite, merging into parts of normal sensibility.

Wounds of the thigh dividing the sciatic nerve completely are so rare that we are compelled to construct the full picture of the consequences of such an injury from a comparison of several cases, not one of which is in itself entirely satisfactory.

Thus, in the case of William B. (No. 57[1]), where the injury was caused by a bullet wound of the nerve in the region of the buttock, muscular paralysis was complete below the knee. But we did not see him until ten months after the injury, and by that time sensation had already begun to improve. Yet an examination of fig. 21 shows the well-marked border on the anterior surface of the leg, and on the inner aspect of the foot, produced by a complete lesion of the great sciatic.

[1] *Vide* Appendix, p. 336.

The full extent of the loss of sensation to light touch, produced by division of the great sciatic, is shown on fig. 22. But here, again, although all the muscles supplied by the great sciatic were absolutely paralysed and the anæsthesia to light touch was of the full extent, sensibility to prick had begun to return on the outer side of the leg.

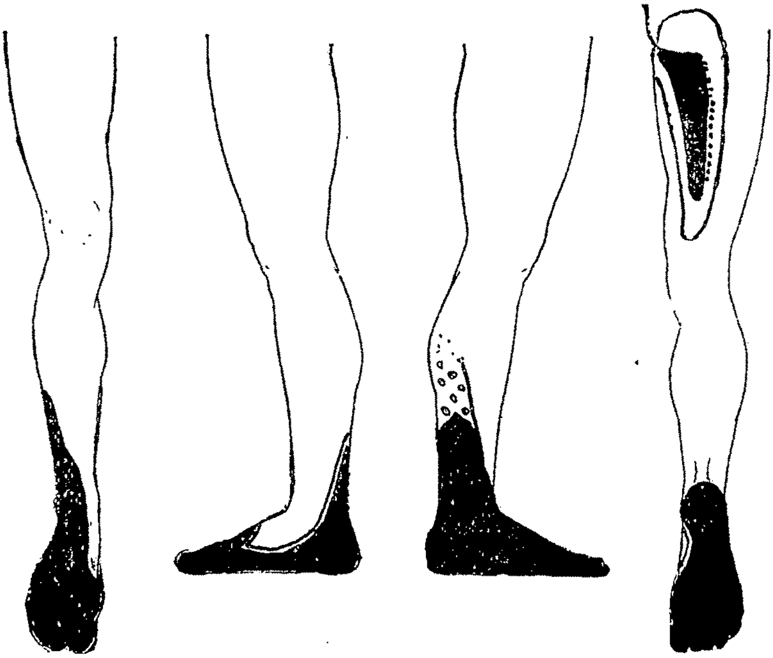


FIG. 21.

To illustrate Case 57. The loss of sensation, produced by injury to the small sciatic, is shown on the thigh and buttock, total insensibility to all cutaneous stimuli being shown in black. The extent of the loss to light touch is shown by a single dark line.

Below the knee the loss of sensation was caused by injury to the great sciatic nerve, which had already begun to recover, the two forms of sensibility returning together, as usual, with partial injuries.

Case 58.—*Complete division of the great sciatic nerve in the thigh.*

On May 29, 1896, Benjamin A. was stabbed in the thigh during a brawl. About the middle of the back of the thigh was an incised wound which had completely divided the semitendinosus muscle. He was collapsed, having lost much blood. The wound was united, after suture of the muscle and healed well.

But it has fallen into disuse as a means of clinical investigation in consequence of the difficulties with which it is surrounded, and the incomprehensible results yielded by the test when carried out in the usual manner. To McDougall (9) we owe a modification which not only makes it easy to use the two points as a test for sensation even in hospital patients, but gives to the records a definiteness otherwise lacking. The blunt points of a pair of compasses are separated from one another for a measured distance. The skin of the affected part is touched, and the patient, whose eyes are covered, is asked to say after each application whether he was touched by one or two points. When they are separated for less than a certain distance, varying with the different parts of the body, the points no longer appear as two on the normal skin. Under the old method the patient was also touched a few times with one point as a test of his trustworthiness. But in all our observations the patient was touched ten times with one point, and ten times with two points, each form of stimulation being applied entirely at random in the series. This modification of the original method adds considerably to the value of the compass test; for we now pay attention not only to the number of times two points are mistaken for one, but also lay stress upon the frequency with which stimulation with one point is said to be two. The results are recorded graphically in the following manner: Every time the patient's answer is correct a stroke is made, above a horizontal line if he was touched with one point, below it if he was touched with two points. An incorrect answer is recorded by a cross. Thus, if he answers "one" when touched with two points, a cross is placed below the line; if one point has been called two, the cross is drawn above the line. A preceding stimulus frequently has an effect upon those which follow it, and to register the order in which the stimuli have been applied is therefore an additional aid to the interpretation of the records. Thus, if the testing began with four double touches correctly answered, four strokes would be ranged below the line from left to right. At the point above the line directly over the last of these double touches would begin the record of the subsequent single stimuli. In this way, the results of all further stimuli are recorded until the number is complete. As ten stimuli of each kind are used, the results can be reduced to percentages at once. For additional accuracy we use the letter "D" whenever the patient is in doubt, and "O" whenever he fails to answer at all.

Perfect appreciation of the compass points at a distance of 2 cm. would be represented thus:—

$$2\text{ cm. }\frac{1 \mid 111 \quad 11 \quad 11 \quad 111}{2 \mid \quad 1111 \quad 111 \quad 111}.$$

If, however, the patient is unable to differentiate the two points at this distance, answering "one" to every stimulation, the record would stand:—

$$2\text{ cm. }\frac{1 \mid \quad 1111 \quad 11 \quad 11 \quad 11}{2 \mid XX \quad XXX \quad X \quad XXXX}$$

Such a formula would show that when 2 cm. apart, the sensation produced by two points is well below the threshold at which discrimination becomes possible. Less complete failure would be represented by some such formula as:—

$$2\text{ cm. }\frac{1 \mid \quad 11XX \quad 1X \quad 1XX1}{2 \mid X1X \quad 1XX \quad XX1}$$

where 50 per cent. of the answers are wrong with one point, 60 per cent. with two points. A curious phenomenon, upon which we shall lay considerable stress, is the tendency to appreciate one point as two over parts of defective sensibility as soon as the limits of accurate discrimination are passed.

In every case our observations were controlled by testing a similar part of the sound hand. We found that almost without exception two points could be accurately discriminated over any part of the normal palm when separated for 1 cm. and applied transversely.

The compasses may be applied either transversely or along the vertical axis of the limb. We have been content in most cases with a strictly transverse position which allows of accurate discrimination at a smaller distance than when the two points are placed in the axis of the limb. But where the area to be examined consists of a long and narrow strip upon the palm, we have also made use of the longitudinal position controlling our observations by an examination of a similar portion of the normal hand.

(Case 28, Table III.) at 6 cm., after the whole palm had become sensitive to prick were erroneous (6 cm. $\frac{1\,|\,10\text{ R.}}{2\,|\,3\text{ R. }7\text{ W.}}$). But six months later, with the points at one-third of this distance apart (2 cm.) an identical formula (2 cm. $\frac{1\,|\,10\text{ R.}}{2\,|\,3\text{ R. }7\text{ W.}}$) was registered over the same area.

Immediately light touch begins to be appreciated over the area affected the compass records respond to the change. At first, sensation becomes uncertain over parts in the neighbourhood of the wrist. A few weeks later light touch can be certainly appreciated over the proximal part of the distribution of the injured nerve, although the distal parts of the palm still remain insensitive. A hand in this condition reveals clearly the close connection between sensibility to light touch and the discrimination of the compass points; near the wrist seven out of ten answers may be right, while over more distal parts of the palm all ten may be incorrect.

Ultimately the whole of the hand becomes sensitive to light touch; but, although the formula shows marked improvement and the distance between the points can be greatly decreased, sensibility to this test still remains defective. This is the stage associated with a "line of change." A pin drawn lightly across the skin from normal to abnormal parts produces a changed sensation as soon as the boundary of the original loss of light touch is passed. The sensation produced becomes more uncomfortable and has a curious tingling which even patients of the meanest intelligence cannot mistake. So long as the hand is in this condition the discrimination of the compass points remains defective, and it would seem that the existence of this state depends on a defect in that form of sensibility which gives precision to the appreciation of two points.

At last sensation may be so completely re-established that the compasses reveal no material difference between the two hands. But so perfect a restoration requires several years, and long before it was reached most patients considered the hand was as good as ever. They ceased to come at our request, or failed to let us know a change of address. A few remained faithful to the end. Amongst them one case of divided ulnar (No. 18, Table II.) gave a perfect formula at 1 cm. exactly two years after the accident, and a case of divided median (No. 4, Table I.) reached the same condition two years and nine months after suture of the nerve.

If anything has happened to retard recovery, if the wound has suppurated or the ends of the divided nerve have not been brought into apposition, compasses may reveal a permanent defect, although the hand has become sensitive to all forms of stimulation. We have seen an old man who accidentally divided his ulnar nerve in 1843. To all forms of touch, to prick and to temperature, sensation had returned. But over the whole distribution of the ulnar nerve sensation was changed, the pin point became sharper, and cotton-wool caused a tingling sensation; compasses were defective over the whole ulnar palm (1 cm. $\frac{1 \mid 9\text{ R. } 1\text{ W.}}{2 \mid 3\text{ R. } 7\text{ W.}}$).

One of our students divided the median nerve of his right hand. When we saw him six years later he responded to all forms of stimulation, but his sensibility to compasses was comparatively low. On the palm of the sound hand he was accurate at 0·5 cm.; on the median area of the injured hand he failed with both one and two points at 1·5 cm. (1·5 cm. $\frac{1 \mid 6\text{ R. } 4\text{ W.}}{2 \mid 3\text{ R. } 7\text{ W.}}$). At the same time he showed an exquisite line of change. In fact, we can state with certainty, that so long as this line of change still remains, the compass points will show that sensation is still below the normal.

When a nerve trunk is injured, but not completely divided, the loss of sensation depends on the gravity of the injury. All forms of sensation may suffer so severely that to all appearances the nerve has been completely severed. But within a month or six weeks sensation to prick may begin to return, and with it the extent of the anæsthesia to light touch and to minor degrees of heat and cold diminishes. This simultaneous return of the two forms of sensibility is a certain indication that the nerve has not been severed, however grave the injury may have been. It is, therefore, of extreme importance to determine with certainty whether sensibility to light touch is returning or not. Cotton-wool is often an untrustworthy stimulus. But the compass points show at once if the loss to light touch has begun to diminish in intensity or extent. For it has been shown that return of sensibility to prick in no way improves the power to appreciate two points; any coincident improvement in sensation shown by the compass test must, therefore, signify a simultaneous improvement in forms of sensibility other than those grouped around sensation to prick. As a matter of fact it will be found, that whenever the compass records show considerable improvement, the parts which have so improved have become sensitive to minor degrees of heat.

Case 29, Table IV., p. 164,[1] is an excellent instance of this condition. A young man cut his wrist whilst loading some bottles upon a van. The loss of sensation produced by the injury corresponded to that which would follow complete division of the median nerve; even the palm was in part insensitive to prick and to the extremes of heat and cold. But exploration of the wound by one of us showed that the nerve was injured without destruction of its continuity. Within a month of the injury sensibility to prick had improved greatly; at the same time we suspected that the parts in the neighbourhood of the thenar eminence were sensitive to cotton-wool. This suspicion was confirmed by the character of the records yielded by the compasses from the palm in the neighbourhood of the thumb and at the base of the index and middle fingers.

Palm at base of thumb, 1 cm. $\begin{array}{l|l} 1 & 9\text{ R. }1\text{ W.} \\ \cline{2-2} 2 & 4\text{ R. }6\text{ W.} \end{array}$.

Palm at base of fingers, 1 cm. $\begin{array}{l|l} 1 & 3\text{ R. }7\text{ W.} \\ \cline{2-2} 2 & 4\text{ R. }6\text{ W.} \end{array}$.

It will be seen that, although the same number of wrong answers were given in both places with two points, the single point was rightly appreciated nine times out of ten over a part where sensation to touch had more distinctly returned, whilst three only out of ten answers were correct over the remainder of the palm. This was no fortuitous difference, for in a month's time the compasses were perfect at 1 cm. over the neighbourhood of the thumb, though still defective over the remainder of the palm (1 cm. $\begin{array}{l|l} 1 & 5\text{ R. }5\text{ W.} \\ \cline{2-2} 2 & 9\text{ R. }1\text{ W.} \end{array}$).

Not infrequently, the pressure of a tight bandage or badly adjusted splint causes a diminution of sensibility to light touch amounting even to complete insensibility to cotton-wool. Such loss is accompanied by a profound alteration in the power of appreciating two points, and even if light touch, tested with cotton-wool, is present, the compasses reveal the diminution in sensibility with unmistakable distinctness.

---

[1] *Vide* Appendix, p. 302.

Case 61.—*Diminution in sensibility to cotton-wool and to the compass test, produced by pressure on the ulnar nerve at the elbow.*

A youth, who was thought to have fractured his clavicle, was strapped by Sayre's method. The little finger became painful during the time the strapping was in position, and later the ulnar half of the hand became numb. When we saw him three months after the accident the muscles of the hand were acting, and contracted normally to the interrupted and to the constant current. Over the whole ulnar area sensation was lowered to cotton-wool, and over the parts where light touch was diminished a prick produced a more intense and diffuse pain than over the normal hand. Water at 25° C. and at 35° C. were everywhere appreciated. At a distance of 1 cm. two points were not discriminated eight times out of ten (1 cm. $\frac{1 | 8\text{ R. } 2\text{ W.}}{2 | 2\text{ R. } 8\text{ W.}}$). Yet two months later light touch had everywhere returned and the record of the compasses at 1 cm. was perfect.

By means of the compasses it is possible to obtain information concerning the relative sensibility of parts which react to all the coarser tests. If the hand is sensitive to all grades of temperature that can be used in practice, and if cotton-wool is appreciated over the whole extent supplied by the injured nerve, it would be impossible without the aid of the compasses to say that one part of this area was more advanced towards recovery than another. But the record of the two points will show that the proximal portion of the palm is more sensitive than that nearer the base of the fingers.

The following case (No. 34, Table V., p. 168[1]) is a good example of the manner in which improvement of sensation can be measured after the hand has become sensitive to all the ordinary stimuli.

A man of 48 years of age cut his left wrist on March 4, 1903. Under an anæsthetic the wound was explored, and several divided tendons were sutured; the ulnar nerve was found to be injured, but not completely divided below the point at which the dorsal branch was given off. This injury caused loss of sensibility to prick over the palmar aspect of the little finger, and anæsthesia to light touch over the ulnar half of the ring finger, the palmar aspect of the little finger and the whole ulnar palm. Over this area intermediate degrees of temperature were not appreciated. The interosseous muscles of the second, third and fourth spaces, and the abductor minimi digiti, were inactive and ceased to respond to the interrupted current. Rapid improvement took place; sensibility to prick began to return, followed quickly by that to light touch. By August 26, 1903, twenty-five weeks after the accident, the hand had become sensitive to all forms of stimulation; but light touch was badly appreciated, and the compasses gave a defective record at 2 cm. The affected parts improved, until just a year after the accident (February 28, 1904) all parts of the palm appeared to be equally sensitive to all forms of stimulation. But over that part nearer to the wrist the compass points gave a record at 1 cm., which showed that the threshold had been approached (1 cm. $\frac{1 \mid 5\text{ R. } 5\text{ W.}}{2 \mid 6\text{ R. } 4\text{ W.}}$). Nearer to the base of the fingers the two points were wrongly appreciated in every instance (1 cm. $\frac{1 \mid 10\text{ R.}}{2 \mid 10\text{ W.}}$). This difference proved that sensibility was steadily returning from the more central to the more peripheral parts of the affected area. On September 25, 1904, eighteen months after the injury, sensation was still changed over the ulnar area; the point of a pin caused more discomfort, and this increased reaction began at the old line of anæsthesia to light touch. Over the proximal parts of the palm at 1 cm. the compasses yielded a somewhat improved record (1 cm. $\frac{1 \mid 10\text{ R.}}{2 \mid 7\text{ R. } 3\text{ W.}}$). More distal portions still showed ten false answers with the two points (1 cm. $\frac{1 \mid 10\text{ R.}}{2 \mid 10\text{ W.}}$).

---

[1] *Vide* Appendix, p. 312.

All the higher forms of sensibility are peculiarly susceptible to the influence of bodily states. We have dwelt upon the way in which recovery may appear to be arrested in consequence of cold or unpropitious weather. But the records yielded by compasses are also influenced by other conditions, such as mental fatigue and particularly alcohol.

By the method we have adopted, an erroneous appreciation of one point is registered equally with the misapprehension of two. And among the formulæ cited to support our contentions in this chapter, the reader will have been struck with the frequency of mistakes in the upper row of symbols. By most clinical observers such errors are supposed to show that the patient is untrustworthy; but experimental psychologists have long recognised that this doubling of a single stimulus is a phenomenon closely associated with an approach to the threshold of sensation. If the compasses are separated for a distance too small for just appreciation, every stimulus, whether by one or two points, is frankly called one. But, if the distance is increased, some stimuli with two points will be called "one," some with one will be called "two." Occasionally every touch with a single point appears to be double, although every touch with two points is correctly appreciated. Such a complete reversal never occurs in our experience with hospital patients, except over parts where the sensibility has been lowered by a definite nerve injury. For however careless, stupid, or alcoholic may have been the subject of our examination, we never observed an unbroken series of doubled single touches amongst the control tests we always applied to the uninjured hand.

Case 5 (Table I. and fig. 3, L.).—*Gradual recovery of sensibility to the compass test after suture of the median nerve.*

On October 2, 1902, a butcher cut his wrist with a knife. Two days later sensation was lost over the area shown in fig. 3, L. The space between the border of the loss to prick and that to light touch was sufficient to permit of the compass points being applied at a distance of 2 cm. apart, and the records were as follows:—

October 4, 1902, 2 cm. $\frac{1 \mid \quad 10\text{ R.}}{2 \mid 4\text{ R. } 6\text{ W.}}$, 1 cm. $\frac{1 \mid 10\text{ R.}}{2 \mid 10\text{ W.}}$.

At the operation on October 4 the median nerve was sutured, and sensibility to prick began to return in the usual way. Thirty-three weeks later (May 24, 1903) light touch appeared to be lost over the whole median area, but the compasses showed slight improvement over the palm.

May 24, 1903, 2 cm. $\frac{1 \mid 7\text{ R. } 3\text{ W.}}{2 \mid \quad 10\text{ R.}}$, 1 cm. $\frac{1 \mid 6\text{ R. } 4\text{ W.}}{2 \mid 8\text{ R. } 2\text{ W.}}$.

By September 23, 1903, light touch was no longer lost over the palm, and the compass records showed material improvement.

September 27, 1903, 2 cm. $\frac{1 \mid 9\text{ R. } 1\text{ W.}}{2 \mid 9\text{ R. } 1\text{ W.}}$, 1 cm. $\frac{1 \mid 4\text{ R. } 6\text{ W.}}{2 \mid \quad 10\text{ R.}}$.

On November 25, 1903, all forms of sensation were appreciated, but a well-defined change occurred at a line corresponding to the border of the previous anæsthesia to light touch. The compasses showed still further improvement in that the record at 2 cm. was now perfect (2 cm. $\frac{1 \mid 10\text{ R.}}{2 \mid 10\text{ R.}}$). At a distance of 1 cm. every stimulation with a single point was called "two," the phenomenon of "double ones" in a perfect form.

	Oct. 4.	May 24.	Sept. 27.	Nov. 25.
2 cm.	$\frac{1 \mid 10\text{ R.}}{2 \mid 4\text{ R. }6\text{ W.}}$	$\frac{1 \mid 7\text{ R. }3\text{ W.}}{2 \mid 10\text{ R.}}$	$\frac{1 \mid 9\text{ R. }1\text{ W.}}{2 \mid 9\text{ R. }1\text{ W.}}$	$\frac{1 \mid 10\text{ R.}}{2 \mid 10\text{ R.}}$
1 cm.	$\frac{1 \mid 10\text{ R.}}{2 \mid 10\text{ W.}}$	$\frac{1 \mid 6\text{ R. }4\text{ W.}}{2 \mid 8\text{ R. }2\text{ W.}}$	$\frac{1 \mid 4\text{ R. }6\text{ W.}}{2 \mid 10\text{ R.}}$	$\frac{1 \mid 10\text{ W.}}{2 \mid 10\text{ R.}}$

The above table shows clearly that recovery of sensibility is accompanied, not only by an increased appreciation of two points, but also by a simultaneous uncertainty in the sensation produced by single stimuli. At any one distance, such as 2 cm., the power of discrimination gradually increases until at last the record of both one and two points may become free from error.

But, in the above instance, when the points were 1 cm. apart every single stimulus was thought to be double, though every double stimulation was rightly appreciated. The serial arrangement of the records at this distance shows the gradual evolution of this phenomenon.

CHAPTER 11.—SENSIBILITY OF THE HAIRS.

Whilst watching the recovery of sensation after division of the ulnar nerve in one of our patients of unusual intelligence, we were astonished at the rapidity with which the back of the hand became sensitive to cotton-wool. We imagined that this was an individual peculiarity, until he gave us the clue, that led us back to a reconsideration of the sensibility of the hairs. When we stimulated with cotton-wool that part of the dorsal surface of the hand supplied by the ulnar nerve, he told us that the sensation radiated widely over the parts affected, and possessed a tingling character foreign to the sensation produced by stimulating normal parts of the hand. Now radiation and a tingling quality had come to be associated in our minds with that form of sensibility which returns early to the affected hand; pain, cold and heat, in as far as they are appreciated by a hand in the first stage of recovery, all produce sensation with this peculiarity. It seemed, then, that on the dorsal surface of the hand some structure had become sensitive to cotton-wool at about the time when sensibility to prick was fully established. Such a structure is to be found in the hairs which are never absent from the back of the hand of men, and are present even on the hands of most women.

Case 15 (*vide* Table II., p. 156, and fig. 1, G, p. 127).—*Complete division of the ulnar nerve at the point where its dorsal branch is given off. Presence of sensation to cotton-wool due to the innervation of the hairs before the return to the hairless skin of sensibility to light touch.*

A lighterman, aged 29, was admitted to the Poplar Hospital on July 29, 1903, with a cut across his left wrist. He had lost sensation over the full ulnar area shown in fig. 1, G, p. 127. The wound was explored and the ulnar nerve was seen to be divided at the point where it gave off its dorsal branch. The upper end, a single trunk, was sutured to the two branches which constituted the peripheral portion of the nerve. All the muscles in the hand supplied by the ulnar nerve were completely paralysed and ultimately lost their reaction to the interrupted current. The wound suppurated and did not heal for seven weeks.

On December 2, 1903, sensibility to prick and to ice had returned over the palm, but was absent over the same area as before on the dorsal surface of the hand, which did not become sensitive to these stimuli until March 23, 1904 (237 days after suture).

By June 12, 1904 (320 days after suture), the whole of the dorsal surface of the hand not only reacted to prick and to ice, but had become sensitive to stimulation with cotton-wool. But when that part of the hand supplied by the dorsal branch of the ulnar nerve was shaved, it became at once entirely insensitive to any form of light touch. This experiment was repeated many times under varying conditions, for it was not until April 9, 1905 (619 days after suture), that this portion of the hand became sensitive to cotton-wool when shaved.

From the beginning, this patient recognised that the sensation caused by cotton-wool over the normal parts of the back of the hand was different from that over the affected area. He said: "When you touch the back of my hand (over the ulnar border) it is a kind of crawling, prickly feeling." Later, he learnt to associate this peculiar sensation so definitely with hairs, that he could tell us if after shaving we had left any still standing above the surface. This explanation received additional support from the fact that sensibility to warmth (below 40° C.) and to the painless interrupted current was absent over the area on the dorsal surface of the hand, which exhibited this radiating and abnormal sensibility to cotton-wool.

A similar condition appeared in one of us during the recovery of the radial half of the back of the hand after experimental division of the radial (ramus superficialis nervi radialis) and external cutaneous nerves in the neighbourhood of the elbow. Within eight months of the operation, this part of the back of the hand had become sensitive to prick, to ice, and to water at 50° C. With this return of sensibility, the hairs began to react to cotton-wool, and this stimulus evoked a curious radiating sensation with a characteristic tingling quality. True localisation was impossible, and the skin over the same parts became, when shaved, entirely insensitive to cotton-wool.[1]

In both these instances recovery was considerably delayed. The wound in the first suppurated badly; in the second case the nerves had been divided at the elbow, and the period between the return of sensibility to prick and that of the recovery of light touch was consequently longer than usual. This return to the hairs of a peculiar form of sensibility characterised by wide radiation seems to occur when the sensation to prick, to ice, and to water at 50° C. has been present alone over the affected parts sufficiently long to be fully restored.

It would seem, then, that return of sensibility to prick and to the more extreme degrees of heat and cold brings a return of sensation to the hairs on the back of the hand. They then react not with that well-localised sensation we are accustomed to associate with stimulation of hairs, but with a wide-spread tingling, analogous to the radiating sensation produced by a prick or by cold water at the same stage of recovery.

---

[1] This case will be dealt with more fully by Dr. Rivers and one of us in a subsequent number of this Journal.

Sensibility is gradually restored, and the back of the hand becomes sensitive to cotton-wool even when shaved. As sensation becomes more perfect the tingling quality disappears, the stimulus can be localised correctly, and the hairs regain their normal sensibility.

We have shown that, under certain conditions, the hairs may regain a peculiar form of sensibility at the time when the affected parts are sensitive only to prick and to the extremes of heat and cold.

Plucking a normal hair will, in most cases, cause pain, and it is this sensibility to pain that returns to the hairs when they react in this manner to stimulation with cotton-wool.

But it is obvious that stimulation of normal hairs produces a well-defined and well-localised sensation differing entirely from the radiation and tingling so characteristic of all sensation at the close of the first stage of recovery. The hairs must, therefore, be endowed with some additional innervation other than that which may be restored with the return of sensibility to prick.

If it were possible to examine a part sensitive to light touch only, the significance of this innervation would become manifest. We have fortunately been able to examine five cases where sensibility to cotton-wool was present over parts insensitive to prick and to the extremes of heat and cold. Here the hairs were in a sensory condition, complementary to that described in the first part of this chapter.

The first instance (Case 85)[1] of this remarkable condition occurred in a plumber who divided the median together with the radial and part of the external cutaneous nerve in the neighbourhood of the wrist. Over the back of the hand in the region of the first interosseous space was a small area entirely insensitive to prick and to the extremes of heat and cold. But over this patch of skin he was sensitive to stimulation with cotton-wool, or to any other stimulus which affected the hairs. Here he also correctly appreciated the difference between water at 25° C. and 38° C.

---

[1] *Vide* Appendix, p. 328.

This was almost exactly the condition of the triangular patch of dissociated sensation which appeared in one of us after experimental division of the radial (ramus superficialis nervi radialis) and external cutaneous nerves at the elbow.

This power of appreciating the movement of hairs, and of localising the point at which the stimulus is applied, can be present although the skin is insensitive to all forms of temperature, and even when, after shaving, it becomes entirely anæsthetic, as shown by the following instance.

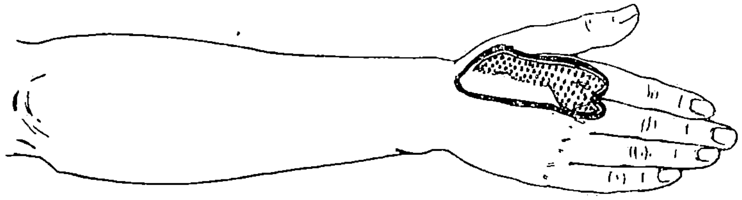


FIG. 27.

To show the extent of the loss of sensation produced by division of the internal branch of the radial and posterior branch of the external cutaneous nerves in Case 62.

The dotted area was insensitive to all forms of cutaneous stimulation. Within the area enclosed by a thick black line the hairs were sensitive, but all sensation of pain, heat, and cold, was absent.

Case 62.—*Division of the internal branch of the radial nerve (ramus dorsalis nervi radialis superficialis), with the posterior branch of the external cutaneous at the wrist, producing an area of dissociated sensibility.*

W. C., aged 21, cut his right wrist with glass on May 18, 1905. The same evening the internal branch of the radial nerve and the tendon of the supinator longus were sutured.

On May 24 we found that he was insensitive to prick over the radial half of the dorsum of the hand (fig. 27.) Sensation was unaltered over the thumb and fingers. The whole of this area was insensitive to all degrees of temperature, but he responded briskly to pressure or any form of deep touch.

Stimulation with cotton-wool was not appreciated over a considerable portion of this area. But over a strip towards its ulnar side, about 4·5 cm. in length and from 1·2 to 1·5 cm. in breadth, cotton-wool produced a tickling sensation. Over the normal parts plucking a hair caused him to say that he was being pricked; but within the area of dissociated sensation he said: "You are touching me, you tickle me." Over the area insensitive to all cutaneous stimuli but sensitive to deep touch, he failed entirely to appreciate a pull sufficiently severe to lead to the removal of the hair.

The sensation produced by cotton-wool within this area of dissociated sensibility was a well-localised tickling, entirely different from the radiating tingling sensation described in the first half of this chapter.

He failed in every case to discriminate two points of the compasses at 4 cm., applied longitudinally over this area; over a similar part of the sound hand the test gave a perfect result at 2·5 cm.

After completing these observations the back of the hand was shaved. The whole of the parts insensitive to prick and to temperature were then found to be entirely anæsthetic to cotton-wool, proving that the sensibility previously existing must have been due to the hairs. Over normal parts of the back of the hand when shaved cotton-wool could be accurately appreciated.

Thus, in conclusion, we believe that the hairs receive a double innervation. When all cutaneous nerves are divided and deep sensibility alone remains a hair can be plucked out without producing any sensation. But as soon as the hand has become fully sensitive to prick and to the extremes of heat and cold, pulling the hairs produces pain, and stimulation with cotton-wool evokes a peculiar radiating, tingling sensation. Later, when the hand has regained sensibility to light touch, this tingling quality disappears, giving place to the well-localised sensation produced when normal hairs are gently moved.

Should the part be sensitive to light touch, though insensitive to prick, movement of the hairs will produce a well-localised tickling sensation. But a hair can be plucked out without producing more than a sensation of touch.

CHAPTER 12.—HYPERALGESIA.

A hand that has passed through the first stage of recovery has become sensitive to prick. So great is the discomfort, and so brisk the movement of withdrawal, when the hand is pricked, that the parts affected are not infrequently said to be "hyperæsthetic" (or more accurately "hyperalgesic"). We have shown that excessive reaction to this form of stimulation is associated with loss of all the finer forms of sensation; it is due to the presence of a peculiar form of sensibility. It is, in fact, the expression of a deficiency rather than of excess, and it disappears gradually with the return of the higher forms of sensation.

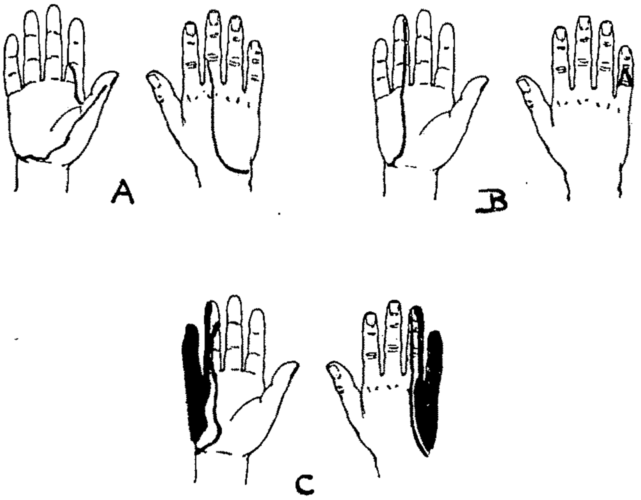


FIG. 28.

A shows the extent of the hyperalgesia produced by irritation of the ulnar nerve in Case 63.

B shows the extent of the loss of sensation to light touch present before the operation.

C shows the loss of sensation produced by dividing the ulnar nerve in the same patient.

But a true exaggeration of sensibility to pain may exist apart from any defect in sensation of light touch. Such hyperalgesia is rarely the result of wounds of peripheral nerves in modern surgical practice, but it underlies the remarkable condition described by Weir-Mitchell (14) under the name of "causalgia" and can still be seen as he described it after injuries particularly with the older forms of bullets. G. H. (Case 63)[1] showed this sensitiveness in so typical a form that a description of his condition might have been taken from the pages of Weir-Mitchell.

He was wounded at Tweefontein on July 22, 1901, by a bullet that entered 4½ ins. (11·5 cm.) below the internal condyle of the humerus, passing across the forearm to the radial side. Not until he had been in hospital three weeks did the hand become painful. The pain became steadily more intense, and when we saw him first (January 26, 1902) was constant. At that time, the skin of the hand was characteristically smooth, glossy and of a pinkish-blue colour, the fingers tapered and the nails were long and curved.

The hand was intensely tender over a large area, occupying the palm, the ulnar half of the thenar eminence, the palmar aspect of the thumb and the palmar aspect of the little, ring and middle fingers. Over the dorsal surface this tenderness occupied the ulnar half of the hand and extended to the tendon of the ring finger. The skin of the dorsal surface of the little, ring and middle fingers was intensely sensitive to pinching, to pressure with the head of a pin and to the pin-point.

Sensation to light touch and to the painless interrupted current was lost over the usual ulnar area, and here the discrimination of the compass points was extremely defective. Sensibility to the more extreme degrees of temperature was perfect everywhere, but ice and water at 50° C. tended to cause pain.

This man's hand, as far as the loss of sensation was concerned, was exactly in the condition which follows an injury without complete destruction of the ulnar nerve. Hitherto in all our cases excessive sensibility to prick has been confined to the so-called anatomical distribution of the affected nerve; that is to say, over-sensitiveness to prick so far has always coincided with the limits of loss to the higher forms of sensation. When the ulnar nerve was destroyed it lay within an area on the palm to the ulnar side of a line through the axis of the ring finger.

---

[1] *Vide* Appendix, p. 316.

But here the tenderness exceeded the area of loss of sensation caused by injury to the nerve, extending on to the median half of the palm. Moreover, it was found over parts that were in no way insensitive to light touch, the interrupted current, or the two points of the compasses.

Obviously this wide extent of hyperalgesia might be explained by supposing that the median nerve had also been affected by the injury. But, apart from the total absence of motor or sensory paralysis pointing to injury of this nerve, this explanation was at once negatived by the results that followed division of the ulnar. On the afternoon of January 30, the ulnar nerve was separated from fibrous tissue, the two ends freshened, and then united by means of a graft. Next morning all pain and tenderness had gone from the hand, and the sensation of the ulnar half was such as might have been expected after total division of the ulnar nerve.

It is therefore certain that the impulses, which produced the true hyperalgesia over the median half of the palm, must have travelled by way of the ulnar nerve. The pain must have been caused by some irritation of the trunk of this nerve; and yet the tenderness far exceeded any area of loss of sensation produced by division of the ulnar nerve.

The following case illustrates a similar hyperalgesia produced by injury of the median. The absence of any exploratory operation and our consequent ignorance of the limits of the loss of sensation that would have been caused by division of the nerve affected make it less convincing; but, in the light of the previous observation, the condition would appear to have been caused by injury of the nerve combined with irritation of its trunk.

J. W. (Case 64),[1] an Imperial Yeoman, was shot through the arm with a Martini bullet on August 1, 1901. The Boers

---

[1] *Vide* Appendix, p. 332.

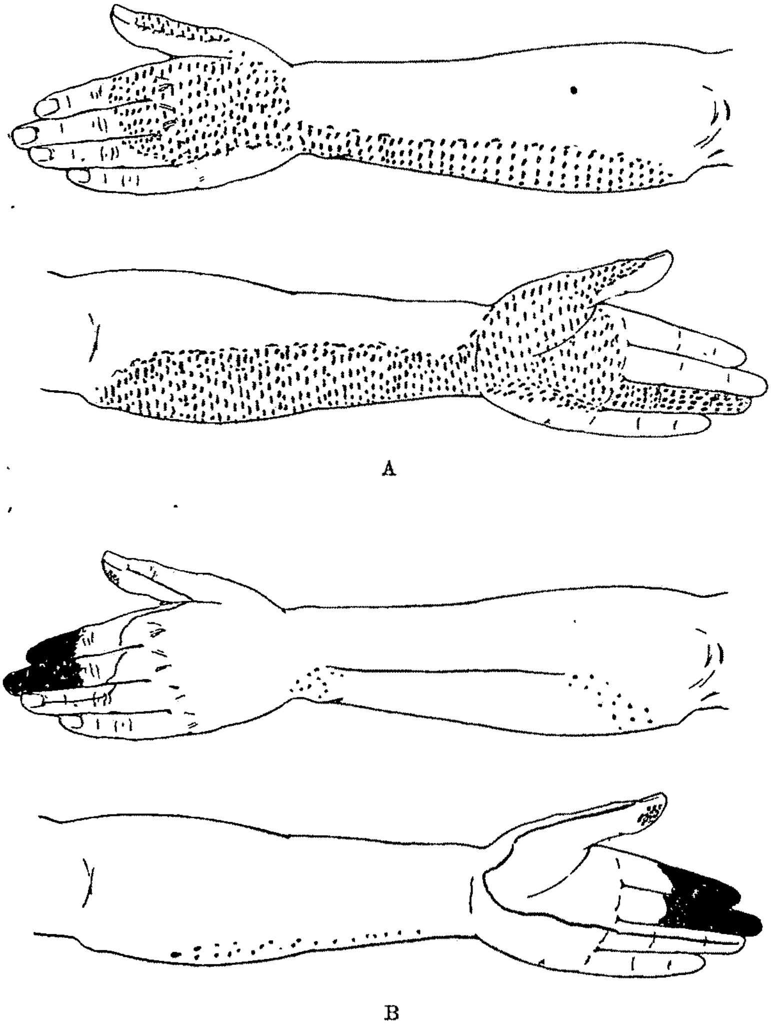


FIG. 29

A, to show the area which became intensely tender in Case 64.
B, to show the extent of the loss of sensation in the same case. Total cutaneous insensibility is shown in black. The area of loss of the higher forms of sensation (light touch, &c.) is enclosed in a single line.

took from him all they wanted, and he lay for two days and one night on the veldt. He was then found and taken to hospital. The wound "became very foul," and did not heal for more than five weeks. At first the arm was painless, but about a month after he was wounded, pain began in the hand and steadily increased. After a while the pain was always present and of about the same intensity, except when the weather was cold. In the winter it was scarcely troublesome so long as the hand was exposed to the cold and cold water always removed the pain for a time.

The bullet had entered the left arm at a point 1½ ins. (4 cm.) above the external condyle of the humerus; 2¾ ins. (7 cm.) above the internal condyle lay the wound of exit, oval in shape and 1 in. (2·5 cm.) in its longest diameter. Both scars showed a tendency to become keloid.

This case was complicated from both motor and sensory aspects by injury to the musculo-spiral nerve. But for the purposes of the present chapter, it will be well to concentrate our attention on the sensory condition of the palm. Injury to the median nerve had produced loss of sensation exactly corresponding to that usually found, when the loss to prick has cleared away from the palm. Light touch and the minor degrees of heat and cold were lost over an area occupying the radial half of the palm, the index, middle, and half the ring fingers. The palmar aspect of the index and middle fingers and of the thumb were insensitive to prick and to the extremes of heat and cold.

At the same time, the opponens and abductor pollicis muscles were paralysed, wasted and inactive to the interrupted current.

This loss both of motion and sensation pointed to an injury amounting to functional interruption of the trunk of the median nerve. But in addition to these paralytic symptoms, J. W. complained of intense tenderness in the palm of the hand. The elbow supported by a sling, he walked about with his hand exposed, terrified lest it should be touched or jarred. The extent of this tenderness could be marked out by pressure with the head of a pin or by dragging the point lightly across the skin from normal to abnormal parts. Comparison of the two figures (fig. 29, A and B) shows that the distribution of the tenderness considerably exceeded the area insensitive to light touch, extending to the ulnar side of the palm and occupying the whole of the ring finger. To the radial side it transgressed the line of anæsthesia to light touch on the thenar eminence and occupied the skin over the dorsal surface of the terminal phalanx of the thumb. The suffering caused, when the skin of the tender area was touched, exceeded anything we have seen in parts that have become over-sensitive to pain as a result of destruction of the higher forms of sensibility.

Thus we must conclude that this man suffered from an injury which interfered with both sensory and motor functions of the median nerve, and at the same time produced an irritative state manifested by tenderness of the palm of the hand of unusually wide distribution. This true hyperalgesia was absent over those parts of the hand which were totally analgesic, and did not invade the little finger or extreme ulnar border of the hand which, as we have seen, become totally analgesic when the ulnar nerve is divided.

In both the examples already given, the hyperalgesia was accompanied by some loss of sensation over the area supplied by the injured nerve. But in the following instance injury to the anterior division of the external cutaneous caused true hyperalgesia uncomplicated by any loss, even of the higher forms of sensation.

L. E. [Case 45, *vide* p. 180] fell on to a china jug, injuring the anterior division of the external cutaneous in the lower third of the forearm. Two weeks later he complained of pain, and the wound was reopened. When we examined him first, fourteen weeks after the accident, he was complaining of pain over an area extending from the position of the scar to the ball of the thumb.

Over the outer aspect of the forearm below the scar was an area, extending on to the thenar eminence, exquisitely tender to any form of pressure or to the point of a pin. Here sensation to cotton-wool was perfect, and two points could be discriminated at a distance of 3 cm. when applied in the longitudinal axis of the limb. To this test there was no material difference between the two forearms.

From these examples it would seem that true hyperalgesia may make its appearance. But it is of rare occurrence and never arises immediately after injury.

It may co-exist with a variable amount of diminution in the sensory functions of the nerve, but disappears if the nerve is divided. It tends to return unless the injured portion is excised and the two ends sutured together. Simple division of the nerve does not suffice to effect a permanent cure.

The area occupied by this hyperalgesia extends to the remotest ramifications of the injured nerve as discovered by anatomical dissection and transcends on all sides the limits of even the most extensive loss of sensation produced when the same nerve is divided. When it occupies parts that are sensitive to light touch, its presence does not disturb the normal distance at which two compass points can be distinguished.

CHAPTER 13.—CHANGES IN THE SKIN ASSOCIATED WITH NERVE INJURIES.

It is a matter of universal experience, that parts within the territory of an injured nerve are liable to undergo changes in the rate and nature of their growth. The nails may grow with unusual rapidity, and the skin become thin and glossy; or the epithelium remains heaped up into masses, and the growth of the nails may be scarcely perceptible. The parts may ulcerate, and painless whitlows be produced, in consequence of trivial injuries. Blisters arise without a burn or other assignable cause.

Such changes fall naturally into two groups, those associated with absence of sensibility to pain, and those that occur in parts that are hyperalgesic and the seat of spontaneous pain. Of these we shall first consider disturbances of nutrition in parts insensitive to pain, not only because they more commonly follow the nerve wounds of civil life, but because they yield data required for a complete understanding of the trophic changes which accompany hyperalgesia.

When one of the nerves of the palm is completely divided the skin no longer desquamates so readily over the area of complete analgesia. This is particularly obvious when the patient happens to be a man who works with his hands. Normal parts of his horny palm protected by the cotton-wool of the dressings tend to become softer with disuse, but over the abnormal area the epithelium is not shed, remaining for many weeks as a rough layer on the surface.

When the wound is healed, and the splint removed from the hand, this want of desquamation is still visible in patients of the hospital class. But, if the hand is well scrubbed with soap and warm water, the dry epithelium comes away in flakes, exposing skin over the affected area, pinkish-blue in colour, colder than normal to the touch. This skin is inelastic and wrinkled, and the normal whorled markings are intersected by innumerable fine lines.

Over the back of the hand, these changes are even better seen than over the palm. Division of the radial and external cutaneous nerves[1] caused the skin of the radial half of the back of the hand to become inelastic; it somewhat resembled the skin of an old man. But in addition to this senile appearance, the superficial layers of the epithelium had formed scales; the affected parts were evidently drier than the normal skin, and the cracks better marked. This gave to the radial half of the hand an appearance resembling the skin of a toad. These changes corresponded exactly with the area insensitive to prick, to heat and to cold. The hairs were very irregular and did not lie in sweeping masses; they stood up or were laid in an uneven manner, each hair occupying a different position, as on an uncombed head. The whole of this area was of a slightly deeper red than the rest of the skin of the hand, and so definite was the difference in colour that it became impressed on the photographic plate as a well-defined patch darker than the surrounding skin.

---

[1] The case of H. H., to be reported in full in a subsequent paper with Dr. Rivers.

In this condition the whole of the area insensitive to prick does not sweat. This causes a dryness which adds greatly to the scaly appearance of the affected part. If a pin is dragged lightly from normal to abnormal parts, it produces over the healthy skin a fine red mark which disappears rapidly, but over parts insensitive to prick, the point produces a white powdery line that lasts for many hours, or even days. This is best seen over the forearm or back of the hand, and is evidently due to the removal of dry epithelial scales.

A prick over an area in this condition draws blood easily, and the marks caused by this stimulation last for many hours after they have faded from the normal skin.

All these abnormalities disappear with the return of sensibility to prick. The hand or other affected part begins again to sweat, and may become moister than the surrounding skin. On the arm and leg, when sensibility to prick has returned, the skin no longer shows any distinctive peculiarity. But over the hand it becomes of a pinkish colour, and the rough hand of a man may gain an almost feminine fineness.

As soon as the hand becomes sensitive to prick, the original sensation of stiffness disappears and is replaced by soreness. It no longer seems to be encased in a tight glove, or to be painted with a layer of collodion; movement now causes pain, as if a bruised part were disturbed.

During the time when the skin is insensitive to pain it is peculiarly liable to injury; a burn or a cut is unperceived and it is therefore neglected. In this way sores and ulcers are produced, which from their situation or from the nature of the infection may lead to the destruction of a considerable portion of the finger. One of our patients, a stonemason, refusing to trouble about a painless ulcer, ground away the terminal phalanx of his finger against the stone he was occupied in sawing. If protected from

TABLE VII.—TROPHIC DISTURBANCES OF THE SKIN

No	Nature of Injury	Nerve United	Nature of Trophic Disturbance
Case 65	Ulnar. Glass cut. Primary suture. Suppurated	Dec. 27, 1898	Blister on ulnar side of palm .. ..
Case 19	Ulnar. Nerve divided and resutured	June 17, 1904	Whole of little finger red and shiny. Large blister
Case 15	Ulnar. Primary suture ..	July 29, 1903	Ulnar side of little finger, ulcer covered with scab
Case 18	Ulnar. Glass cut. Sutured fourteen days later	July 31, 1902	Dec. 3, 1902.—Sore on dorsum of first phalanx little finger
Case 30	Median incomplete. Glass cut	July 31, 1904	Oct. 7.—Small blister on tip of middle finger. Oct. 22.—Irritated to a large sore
Case 29	Median, incomplete. Glass cut, Sept. 20, 1902	Explored same day	Nov. 26.—Blisters on index and middle fingers ..
Case 31	Median, incomplete. Dec. 18, 1904. Glass cut	.. ..	Feb. 15, 1905.—Blister on index .. .. ..
Case 13	Median. Secondary suture	Feb. 6, 1905	Feb. 22.—Blisters between index and middle and middle and ring fingers
Case 12	Median. Secondary suture	Feb. 22, 1904	June 8.—Blister on palmar surface of terminal phalanx middle finger
Case 6	Median. Razor cut. Primary suture In consequence of a stitch abscess sensation became lost again	Oct. 3, 1903	Jan. 10, 1904.—Blisters on tip of index and middle fingers Oct. 26, 1904.—Oval ulcer, came as a blister in night on terminal phalanx middle finger. Dec. 22, 1904.—Large blister over index
Case 7	Median. Glass cut. Suture eight days later	Sept. 14, 1902	Nov. 26, 1902.—Blister on index. Second "blood blister" on same site a few days later
Case 66	Median, incomplete. Glass cut, Aug., 1901	.. ..	Nov. 4, 1901.—Tips of index and middle fingers bulbous. Both nails gone; nail-beds granulating surfaces
Case 5	Median. Knife cut. Operation two days later	Oct. 4, 1902	Dec. 10, 1902.—Ulcer and blisters index. Dec. 30.—Many fresh ulcers. March 8, 1903.—Black blisters on tip of index
Case 8	Median. Wrist cut with a piece of coal, Feb. 7, 1903	Feb. 9, 1903	March 4, 1903.—Two ulcers and a blister on index. Blister on palmar surface of middle finger. May 13.—Whole terminal phalanx of middle finger an open sore. Nail gone
Case 85	Median with radial and part of external cutaneous. Glass cut, April, 29, 1902. Secondary suture	June 3, 1902	Aug. 14.—Many ulcers and blisters on thumb, index and middle fingers. Sores continued to appear and tip of index became disorganised

FOLLOWING DIVISION OF THE NERVES OF THE HAND.

Cause Assigned	Result	Condition of Sensation at Time of Appearance of Tropic Change
Burn	Healed well	Protopathic loss over little finger and ulnar border of hand.
None	Healed in one month	Protopathic loss over little finger and ulnar border of hand.
Burn, Nov. 18	Healed in eight weeks	Protopathic loss over little finger, but recovered on palm.
Rubbing the affected parts	Healed Dec. 31 ..	Protopathic clearing rapidly. Loss over little finger. Cleared Jan. 14, 1903.
No definite cause. Sore irritated with oxalic acid	Healed with return of sensation Dec 14	Protopathic loss had begun to clear and soon corresponded almost exactly with the large blister. Protopathic cleared Dec. 14.
Driving..	Healed by Feb. 11, 1903	Oct. 22.—Both forms of sensation began to clear together. Protopathic loss over last two phalanges middle and ring fingers on Nov. 26.
Burn with cigarette ..	Healed by March 1 ..	Protopathic loss index and middle fingers. Had begun to clear rapidly.
None	Healed by March 8 ..	Protopathic loss middle and index fingers.
Pinched finger ..	Healed by July 13 ..	Protopathic loss two terminal phalanges index and middle fingers. Cleared Aug. 28.
None	Healed by March 30 ..	Protopathic loss on index and middle fingers. Not clearing. Cleared by April 27.
None	Healed by Nov. 16.	
?	Healed by Jan. 7, 1903	Protopathic loss over two terminal phalanges of middle finger. Clearing rapidly. Then remained stationary throughout winter. Cleared June, 1903.
.. ..	Nails began to reform Jan., 1902	Protopathic loss over two terminal phalanges index and middle fingers. Cleared May, 1902.
Work as a butcher. No cause known	Healed Dec. 30. No trophic changes after May, 1903	Protopathic loss index and middle fingers. Began to clear March, 1903. Cleared May 24.
All said to be due to burns. These healed, but the finger broke out again when he began to clean pewter with sand and soda water	.. ..	Protopathic loss had begun to clear from palm on March 4, but index and middle fingers were insensitive. May 13, protopathic sensibility lost over one and a half terminal phalanges of middle finger.
Burns during work as a plumber	All healed soundly by Dec. 20, 1903	Protopathic lost completely over thumb, index and middle fingers. Protopathic sensibility returned completely Dec. 20, 1903.

No	Nature of Injury	Nerve United	Nature of Trophic Disturbance
Case 11	Median. Glass cut, May, 1901	May 16, 1902	On July 15, 1901, ulcers were present on thumb, index and middle fingers. Began as blisters; showed no signs of healing. Fresh sores appeared and by April, 1902, two terminal phalanges index and middle fingers, were enlarged, and tip of index was destroyed. No ulcers during two months out of work. Oct.—Middle finger amputation for necrosis of bone. Dec. 21, 1902. Fresh ulcer
Case 10	Median. Glass cut. Primary suture	Jan. 21, 1905	March 15.—Blister on index. Cut on tip of index has formed a small sore. April 19.—Whole tip of finger ulcerated. Terminal two phalanges involved in a large blister
Case 28	Median and ulnar. Glass cut, Sept. 24, 1902	April 17, 1903	March, 1903.—Tips of fingers grew hard and discharged. Placed in hot water they became blistered. April 16.—Nail destroyed; granulating sore. Whole terminal phalanx of index enlarged
Case 60	Complete ulnar, partial median. Glass cut. Primary suture	Nov. 30, 1904	Jan. 14, 1905.—Two blisters on dorsum of little and ring fingers. Jan. 26.—Further blisters appeared
Case 25	Median and ulnar. Glass cut. Primary suture	Dec. 24, 1902	March 8, 1903.—Dorsal surface second phalanx middle and ring fingers scabbed ulcer
Case 67	Partial median and ulnar. Cut wrist with penknife, Oct. 21, 1903. Secondary suture of ulnar	Jan. 11, 1904	Dec. 21, 1903.—Blisters on middle and ring fingers. None elsewhere. These formed ulcers
Case 26	Median and ulnar. Cut with jug. Primary suture	Oct. 26, 1902	March 4.—On tip of index a brown callosity, and on middle finger a blister

injury and infection, these sores heal completely; but comparatively trivial injury will cause them to break out again. The healing differs from that of an ulcer on normal parts only in the slowness of the process. Blisters are liable to form a callosity, which if removed leaves a raw surface. From such a sore, especially when situated in the loose skin on the back of the hand, blood and serum can be expressed by manipulations, insufficient to cause such exudate from a sore healing under normal conditions. The defective elasticity of the skin will cause these sores

Cause Assigned	Result	Condition of Sensation at Time of Appearance of Trophic Change
Vorked as a stone sawyer	Jan. 25, 1903, all sores healed, and in spite of work none have appeared since	Protopathic sensibility returned Jan. 25, 1903.
ue to using a knife. pril 19. Attributed to hot water	.. ..	Protopathic loss clearing rapidly April 19, 1905.
Vork as a carpenter ..	All sores healed after operation, April 17, 1903. Then broke out again and lasted until Dec. 20, 1903	Protopathic sensation began to return by April, 1903. Finally returned Dec. 20, 1903.
.. .. ..	All healed March 8, 1905	Protopathic sensation began to return to the palm on Feb. 1, 1905.
urn .. .. .,	Healed by July, 1903	Protopathic sensation returned completely July 12, 1903.
ttributed to hot water into which whole hand was dipped	.. ..	Middle and ring fingers only were analgesic. Protopathic sensibility returned rapidly after secondary suture, but had begun to clear before the operation.
.. .. ..	Healed by June, 1903	March 4.—Protopathic sensibility showed first sign of returning. Completely returned August, 1903.

to reopen, and a sore on the back of the hand broke open when apparently healed, in consequence of the stretching caused by gripping firmly the handle of a bicycle.

If the patient is engaged on any work that injures his hand, even slightly, these injuries will lead to the formation of ulcers that may necessitate the removal of the finger. A potman who had divided his median nerve cleaned the pewters with silver sand and soda water; the terminal phalanx of the middle finger became an open sore, the matrix of the nail necrosed, and the nail was lost. Another patient, a plumber by trade, repeatedly burnt the affected fingers in the act of soldering.

But ulcers may make their appearance, when the hand is insensitive to prick, apart from any recognisable injury. Such ulcers always start as blisters which break. Sometimes the contents are serous, but they may be blood-stained and form the so-called "blood blister." Such blisters usually arise at a time when sensibility to prick is beginning to return, but the analgesia has not disappeared from the parts over which the blister is situated.

Whatever their cause, whether they arise from external injury or not, all these defects of nutrition disappear with the return of sensibility to pain, and to the more extreme forms of heat and cold. In the case of a plumber (Case 85)[1] a series of ulcers caused by trivial injuries in his trade had led to destruction of the top of his index finger. The whole of the index and middle fingers except the terminal phalanges had become sensitive to prick by October 25, 1903, and all but the ulcer over the terminal phalanx of the middle finger had healed. By December 20, 1903, the whole hand had become sensitive to painful stimulation, and not only had all the sores healed, but no others made their appearance, although the median area of the hand was still insensitive to light touch and to minor degrees of temperature. This immunity from ulceration existed in spite of work continued under unaltered conditions. Similarly a carpenter who had divided both median and ulnar (Case 28, Table III., p. 160)[2] suffered before and after secondary suture of the nerves from ulcers, caused by burns or by blisters arising from the use of his tools. They troubled him up till the complete return of sensibility to prick, eight months after suture, when they healed firmly and never recurred. In April, 1905, the whole palm and fingers were insensitive to light touch and to minor degrees of temperature; but, whenever he burnt his hand the blister healed as quickly and firmly as on the normal side.

---

[1] *Vide* Appendix, p. 328.
[2] *Ibid.*, p. 321.

In this connection Case 6 (Table I., p. 152)[1] is of interest. On October 3, 1903, he divided the median nerve of his right hand, together with most of the tendons on the front of his wrist. The divided tendons and nerve were united next day and the wound healed. In January, 1904, blisters appeared on the termination of the index and middle fingers, said to be caused by burns; those on the radial side of the index finger formed shallow ulcers, which did not heal until April, 1904. By April 27, sensibility to prick and to the extremes of temperature had returned to all parts of the hand, but the whole median area of the palm remained insensitive to light touch. He steadily improved until the end of August, when a swelling appeared at the site of the original wound. This was opened and pus evacuated, probably due to infection of one of the silk sutures, by which the nerve had been united. This suppuration threw back the recovery of the hand to such an extent that the two terminal phalanges of the middle and ring fingers became again insensitive to all degrees of temperature and totally analgesic. On October 23, on waking in the morning, he noticed a blister on the terminal phalanx of the middle finger. Excepting that it was entirely insensitive, this finger was not affected the night before when he went to bed. On October 31 the site of the wound was explored by one of us and the nerve was found to be embedded in a mass of fibrous tissue; it was freed and in a month sensation had begun to improve. But on December 18, when he woke in the morning a large blister had appeared on the dorsal aspect of the terminal phalanx of the index finger. This formed a sore, which did not heal until July 1905. By this date, all analgesia had disappeared and the affected parts had become sensitive to prick and to the extremes of heat and cold, but remained insensitive to light touch. From this time no further blisters have made their appearance.

The distribution of these ulcers is a further proof of the close association between trophic defects and the absence of sensibility to pain. Under no circumstances, unless complicated by acute sepsis, do they extend beyond the analgesic area. A youth, aged 14 (Case 30, Table IV., p. 164), cut his wrist with broken glass, injuring, but not completely dividing, his median nerve (July 31, 1904). Sensation to prick began to return in September, and by October only the terminal phalanges of the middle and index fingers remained insensitive. On October 12, the skin over the tip of the middle finger broke, forming a sore. On October 22, he cleaned the brass of some bedsteads, using oxalic acid for the purpose. When we saw him four days later the skin over the whole terminal phalanx of the middle finger was raised to form a large blister. This occupied the whole analgesic area and was strictly limited to the parts insensitive to pain. At this time sensibility was returning rapidly and the raw surface healed steadily, keeping pace with the return of sensation. By December 14, it had healed completely and the middle finger had become sensitive to the tip.

---

[1] *Vide* Appendix, p. 300.

That the neuralgia consequent on injury to a nerve may be associated with changes in the skin has been known for nearly a century. In 1813, Alexander Denmark (4) reported the case of a man wounded at the storming of Badajos. The bullet entered 1½ ins. above the inner condyle of the humerus and came out on the outer side, in front of the elbow-joint. No date is given for the onset of the pain, which was intense. "I always found him with the forearm bent and in the supine position, supported by the firm grasp of the other hand. . . ." He described the sensation of pain as beginning at the extremities of the thumb and all the fingers except the little one, and extending up the arm to the part wounded. "It was of a burning nature and so violent as to cause a continual perspiration from his face. He had an excoriation on the palm from which exuded an ichorous discharge."

Although this is an excellent description of the condition so fully described by Weir-Mitchell, Morehouse and Keen (14), under the name of causalgia, Denmark makes no mention of the glossy skin that so commonly accompanies this form of hyperalgesia. The first description of this condition was given by Hamilton (5) in 1838. He described the pain and tenderness which may follow nerve injuries, and states that they may be accompanied by redness and swelling resembling the appearance of the skin in inflammation of the fascia or a deep collection of matter.

The first complete account of this state occurs in a clinical lecture by Paget (10) delivered in 1864. As some want of apprehension of his teaching has led to subsequent misunderstanding we give his summary in full.

"Glossy fingers appear to be a sign of peculiarly impaired nutrition and circulation due to the injury of nerves. They are not observed in all cases of injured nerves and I cannot tell what are the peculiar conditions of the cases in which they are found; but they are a very notable sign and are always associated, I think, with distressing and hardly manageable pain and disability. In well-marked cases the fingers which are affected are usually tapering, smooth, hairless, almost devoid of wrinkles, glossy pink or ruddy, or blotched, as if with permanent chilblains. They are commonly also very painful, especially on motion, and pain often extends from them up the arm. In most of the cases this condition of the fingers is attended with very distinct neuralgia both in them and in the whole arm, and its relation to disturbance of the nervous condition of the part is, moreover, indicated by its occasional occurrence in cases where neuralgia continues after an attack of shingles affecting the arm."

To Weir-Mitchell, Morehouse and Keen is due the credit of a complete description of a series of cases illustrating this condition. This they published in 1864, in a volume long out of print, but the original description is quoted and amplified by Weir-Mitchell (14) in his book on the "Injuries of Nerves." He says: "The skin affected in these cases was deep red or mottled, or red and pale in patches. The epithelium appeared to have been partially lost, so that the cutis was exposed in places. The subcuticular tissues were nearly all shrunken, and where the palm alone was attacked the part so diseased seemed to be a little depressed and firmer and less elastic than common. In the fingers there were often cracks in the altered skin and the integuments presented the appearance of being tightly drawn over the subjacent tissues. The surface of all the affected parts was glossy and shining, as though it had been skilfully varnished. Nothing more curious than these red and shining tissues can be conceived of. In most of them the part was devoid of wrinkles and perfectly free from hair. Mr. Paget's comparison of chilblains is one we often used to describe these appearances; but in some instances we have been more strikingly reminded of the characters of certain large thin and polished scars."

In recent years this condition so accurately described by Paget and Weir-Mitchell, and by them associated correctly with pain and tenderness, has been confused with the atrophic conditions that accompany loss of sensibility. Some writers, by the statement that glossy skin is not associated with pain and tenderness, show that they have failed to recognise the essential difference between the condition described by Paget and Weir-Mitchell and the atrophic skin which not infrequently results from division of a peripheral nerve. This confusion is fatal to a comprehension of that condition to which the name "glossy skin" can alone be applied with propriety.

When considering in a previous chapter (p. 245) the hyperalgesia which may follow a nerve injury, we quoted the case of a gunshot wound of the ulnar nerve (Case 63).[1] The whole palm of the affected hand was of a pinkish colour and smooth. The markings were not absent, but the injured hand appeared as if seen through a layer of collodion. Over the dorsal surface of the fingers, particularly over the last two phalanges, the skin was thin and shiny, and the hairs had disappeared. This glossy appearance occupied the thumb, index, middle, ring and little fingers on their palmar aspect and the dorsal surface of the two terminal phalanges of all four fingers.

The palm was intensely tender and hyperalgesia was found over the palmar aspect of the little, ring and middle fingers. On the dorsal surface, the ulnar half of the back

[1] Reported in full in the Appendix, p. 316.

of the hand, together with the whole of the little and ring and the greater part of the middle finger, was intensely tender to pressure or to the point of a pin.

The skin was glossy over an extent somewhat wider than that of the hyperalgesia, but both greatly exceeded the area which subsequently became insensitive after the nerve had been divided (fig. 28, A and C, p. 245).

The characteristic condition of the nails will be dealt with in the next chapter; they were curved and exquisitely tender. On January 30, 1902, the ulnar nerve was dissected at the site of the injury, the two ends freshened and united by a graft. On February 4, the glossy appearance of the skin and all hyperalgesia had disappeared, the sensory state of the hand being that which follows complete division of the ulnar nerve.

True hyperalgesia may exist without the skin becoming glossy, and of the remaining instances cited in Chapter 12, to illustrate this form of tenderness, none were accompanied by this characteristic change. Its full significance can only be determined by an examination of a series of cases, an opportunity that is not likely to occur in civil practice.

We have already dealt with the blisters which are liable to appear over parts totally insensitive to cutaneous stimuli; Weir-Mitchell describes the various forms of eruption that can accompany hyperalgesia. He says: "It was somewhat rare to see any case of glossy skin, especially with causalgia, unattended with vesicles." But in the only complete instance of this condition which has come under our notice, no rash had been present at any time. In one patient who suffered with true hyperalgesia (Case 64)[1] a herpetiform rash was said to have appeared over the little and ring fingers, and ulnar half of the palm, four months before we first saw him.

CHAPTER 14.—CHANGES IN THE NAILS ASSOCIATED WITH NERVE INJURIES.

Since Weir-Mitchell first described systematically the changes in the nails which follow nerve injuries, most observers have contented themselves with supporting or qualifying his statements.

---

[1] Reported in full in the Appendix, p. 332.

No one can doubt that the growth and texture of the nails is profoundly affected when a nerve to the hand is injured, but no systematic observations have been instituted to discover the cause of these changes. The general acceptance, overtly or by implication, of some trophic influence exercised by nerves on epithelial structures has led to an absence of that rigid series of control experiments which are necessary, before any such theory can be upheld.

A nerve injury affecting the hand produces a combination of extremely complex conditions. To ensure union of the divided nerve, the arm is placed upon a splint. The muscles of the hand may be paralysed and useless for many months, so that all those movements necessitated by daily life are materially restricted. Division of one or more arteries at the time of the accident may diminish the supply of blood to the hand, and vasomotor changes may result from the nerve injury. Moreover, the fact that nerve influence has been removed from the hand renders that part increasingly sensitive to the vascular influence of cold; it will become blue at temperatures that produce no such effect upon the normal skin. To correct these bad effects the hand is not uncommonly massaged, and we shall show that this also has a material influence on the growth of the nails.

We tried many methods for registering the growth of the nails, but returned to that originally recommended by Weir-Mitchell. A mark is made with nitric acid on that part of the base of the nail which has just emerged from the cover of the skin; week by week we register the passage of this orange streak until it reaches the free edge and is removed by the scissors. Care must be taken that the acid does not excoriate the nails but acts only as an indelible stain. This is not always easy to carry out in practice, in consequence of the different texture of the nails of the sound and affected hands. If the nails are rough, an application which scarcely stains the normal nail will burn those that are abnormal; or the density and firmness, noticeable in nails in which growth has been long delayed, may render staining difficult without repeated application. After the acid has been placed upon the nails, it is well to wait until the stain begins to appear; then the hand should be well washed and dried before the patient is dismissed, to make certain that the acid does not continue to act harmfully.

Immobilisation of the hand upon a splint retards profoundly the growth of the nails. After a fortnight, the nails on the free hand may have grown to three times the extent of those on the hand that was restrained. These changes are so startling, and have so completely failed to attract attention, that we give shortly the details of some of our most satisfactory observations. It will be seen that the

TABLE VIII.

No.	Age	Sex	Fracture	Days in Splints	Growth of Nails	
					Affected Side	Normal Side
Case 68	57	M.	Right Colles'	14	1 mm.	3 mm.
Case 69	52	M.	Right Colles'	14	0	1·5, 1·0, 0·5, 0·5, 0·5 mm.
Case 70	33	M.	Right radius	14	1 mm.	2 mm.
Case 71	16	M.	Left radius	21	2·5 mm.	3·5 mm.
Case 72	12	M.	Left radius	14	0·5 mm.	2 mm.
Case 73	9	F.	Right ulnar	21	2 mm.	3·5 mm.
Case 74	48	M.	Left radius	28	0	2·5 mm.
Case 75	9	F.	Right radius and ulnar	14	0·5 mm.	1·5 mm.
Case 76	9	M.	Left radius and ulnar	14	0	1·5 mm.
Case 77	10	M.	Separated lower epiphysis of left humerus	14	1·5 mm.	2·5 mm.
Case 78	7	M.	Separated lower epiphysis of left humerus	21	2 mm.	3 mm.
Case 79	13	M.	Shaft of left humerus	28	3 mm.	4 mm.
Case 80	54	M.	Shaft of left humerus	28	0	3 mm.

On this table one number only is given, whenever the nails on all the fingers grew to the same amount. But if they grew differently on any of the fingers, the measure of their growth is given in a series beginning with that of the thumb.

patients were of diverse ages, and the injuries for which the arm was placed in the splint ranged from fracture of the humerus to fracture of the lower end of the radius. In no case was the growth on the two sides even approximately equal, and the uniformity of growth in all the nails of the quiescent hand was remarkable. This diminution of growth is not due to any change in the blood supply of the arm, produced by either bandages or splints, for if the arm is bandaged to a splint, but the hand left free, growth is not materially retarded.

In No. 75, in consequence of fracture of the right radius and ulnar, the arm was placed on a splint with the hand immobilised for fourteen days; in this time the nails had grown three times as much on the left as on the right hand. For the next twelve days the right arm remained on the splint but the hand was freed, and the patient was encouraged to move the fingers. Growth was now equal in the nails of both hands (1·5 mm.). For this reason a sling makes no material difference to the growth of the nails, provided that it is not used in consequence of some affection tending to cause restriction in the movements of the hand. A boy, aged 9 (Case 76), fractured his left radius and ulnar; he was put into splints for fourteen days, during which time the nails of the left hand did not grow to any measurable amount. During the next eight days the arm was kept in a sling, but the nails grew equally on both hands.

Massage causes little definite increase in the growth of the nails of a hand in ordinary daily use. A healthy young woman who had no daily occupation attended the massage department regularly from August 10 to September 21. Her left hand was rubbed for twenty minutes, three times a week, but it is impossible to say that this treatment made any perceptible difference to the growth of her nails.

Forty-six Days without Massage.

	Left.	*Right.*
Thumb	5 mm.	5 mm.
Index	5 mm.	5 mm.
Middle	5 mm.	6 mm.
Ring...	5·5 mm.	6 mm.
Little	5 mm.	5 mm.

Forty-two Days, Left Hand Massaged Three Times a Week.

	Left.	*Right.*
Thumb	6 mm.	7 mm.
Index	6 mm.	6 mm.
Middle	6 mm.	6 mm.
Ring	6 mm.	6 mm.
Little	5 mm.	6 mm.

But if the hand is protected from the cold and rubbed repeatedly, the nails may grow excessively, even though the arm remain in a sling. Of this, Case 80 is an excellent instance. This man fractured his left humerus, and during the twenty-eight days his arm remained in splints the nails of his left hand did not grow appreciably. During the following fortnight, the left arm was kept in a sling, but the hand and arm were massaged every second day. At the end of fourteen days the growth was as follows:—

	Left (affected).	*Right (sound).*
Thumb	2 mm.	1 mm.
Index	4 mm.	0·5 mm.
Middle	4 mm.	0·5 mm.
Ring	4 mm.	1 mm.
Little	3 mm.	0·5 mm.

Throughout the next two weeks he wore no sling, and the hand was rubbed five times only.

	Left (affected).	*Right (sound).*
Thumb	2 mm.	1·5 mm.
Index	1 mm.	2 mm.
Middle	1 mm.	2 mm.
Ring	1 mm.	1·5 mm.
Little	1·5 mm.	2 mm.

In this instance, the return of the hand to its ordinary uses threw back the growth of the nails to a figure on the whole slightly below that of the normal hand. This is an unusual experience; more commonly, the nails of the two hands grow equally if the hand is kept in a sling and massaged every second day.

Thus, whilst immobilisation of the hand on a splint measurably retards the growth of the nails, massage does not universally produce a corresponding increase, provided the hand be normal.

But, when the hand is paralysed or incapacitated from sharing to the usual extent in the necessary movements of daily life, regular massage tends to prevent the extreme retardation of growth that would otherwise occur, and the nails may grow only slightly less rapidly than those of the normal hand.

Muscular paralysis alone will greatly retard the growth of the nails, and probably for this reason irregularity of growth is so manifest a consequence of division of the ulnar nerve. This we had hoped to show from cases of motor paralysis affecting the hand due to destruction of the anterior horns of the spinal cord. But instances where anterior poliomyelitis has affected one hand only so gravely that all movement has been destroyed, are uncommon; the following case shows how considerable may be the retardation of growth from this cause. A child of four years old became suddenly ill in July, 1902, and four days later the right arm and hand were found to be useless.

In February, 1903, she showed all the signs of the paralysis due to anterior poliomyelitis. The right arm was flaccid; the muscles of the shoulder were not acting, with the exception of the upper part of the trapezius. She could slightly extend the middle and ring fingers, but could make no other movement of the hand, forearm or arm. During the summer the hand was neither blue nor cold, but throughout the winter months it was constantly somewhat colder to touch than the normal hand. All forms of sensation were perfect, including the sense of passive position. From February 11 to May 13, a period of ninety-one days, the growth of the nails was almost twice as great on the sound as on the paralysed hand.

	Right (affected).	*Left (sound).*
Thumb...	5 mm.	8 mm.
Index ...	5 mm.	8 mm
Middle...	5 mm.	9 mm.
Ring ...	5 mm.	9 mm.
Little ...	4 mm.	7 mm.

During seventy-seven days (from November 4 to January 20) the growth maintained almost exactly the same proportion, showing that the defect was not due to coldness of the hand.

	Right (affected).	*Left (sound).*
Thumb	5 mm.	7 mm.
Index	4 mm.	7 mm.
Middle	4·5 mm.	7 mm.
Ring	4·5 mm.	7 mm.
Little	4 mm.	5·5 mm.

Whenever we marked the nails of the affected hand we also marked those on the sound side. By this means we gradually accumulated a large number of observations on the growth of normal nails extending, in seventeen cases, over a period of more than a year. Several of these patients remained under observation consecutively for three years. We noticed that the rate of growth differed considerably from time to time, and expected to find that this variation was coincident with the seasons of the year. In a few instances it certainly seemed that the nails grew more rapidly between May and July, and more slowly between November and March. But of the whole seventeen cases only six came within this category, and since normally the growth of the nails is liable to inexplicable variations, we do not consider that our observations are sufficient to establish any such general rule.

So far we have examined only the result of immobilisation and of other conditions acting on a hand whose nerves were uninjured and have shown that limitation of movement is a potent influence in retarding the growth of normal nails.

Turning to cases of complete division of one or more nerves of the hand, we find that want of movement is also the prime factor in the profoundly altered growth of the nails which follows any lesion causing paralysis.

Division of a sensory nerve alone produces no change in the growth of the nails that spring from the fingers which have become completely insensitive.

Case 81.—*Division of the digital branches of the median and ulnar nerves supplying the ring finger. Total loss of cutaneous sensibility over the two terminal phalanges. Absence of any alteration in the growth of the nail of the affected finger.*

On October 12, 1903, whilst working as a cabinet maker, G. W. cut the palm of his hand with a chisel. He came the same night to the London Hospital and two tendons were sutured; the wound is said to have healed well.

When he first came under our notice on December 16, 1903, a longitudinal scar ran from the head of the fourth metacarpal bone to ½ in. (about 1·2 cm.) below the fold of the wrist. This scar, about 2½ ins. (6·5 cm.) in length, was crossed somewhat obliquely at about its centre by a second smaller scar.

None of the intrinsic muscles of the hand were paralysed or wasted and the tendon of the flexor sublimis that went to the ring finger had united perfectly.

From the moment of the accident, he recognised that the ring finger was numb. The whole of the skin over the two terminal phalanges we found to be insensitive to prick and to all forms of heat and cold; the area of insensibility to light touch corresponded on the dorsal surface of the finger to this analgesia but extended on the palmar aspect as far as its base.

A rounded ulcer with thickened edges and a smooth granulating floor was situated over the palmar aspect of the terminal phalanx of the ring finger, evidently healing slowly.

For forty-two days the condition of sensation did not change materially. In this time the nails grew but slightly less on the affected finger than on that of the sound hand. Moreover, this small difference was found in all the four fingers and can be accounted for most probably by some want of use.

	Left (*affected*).	*Right* (*sound*).
Thumb	4·5 mm.	4·5 mm.
Index	4·5 mm.	5 mm.
Middle	4 mm.	4·5 mm.
Ring	4 mm.	4·5 mm.
Little	3 mm.	3·5 mm.

In the case of one of us, where the radial and external cutaneous nerves were divided at the elbow, the nail of the thumb grew to exactly the same extent on the two sides, although the nail-bed and dorsal surface of the thumb were insensitive to all cutaneous stimulation.

Division of the median nerve at the wrist without injury to the tendons is an occasional accident. In such a case it will be found that the nails grow equally on the two sides. Thus after suture of the divided median nerve, A. C. (Case 4, Table I., p. 152) was kept for thirty-five days on splints. During this time the nails grew to the following amount:—

	(*Left affected*).	*Right* (*sound*).
Thumb	1 mm.	5·5 mm.
Index	1·5 mm.	5·5 mm.
Middle	1 mm.	5 mm.
Ring	2·5 mm.	5 mm.
Little	3 mm.	5·5 mm.

But in the thirty-nine days which followed they grew equally, in spite of the absolute loss of cutaneous sensibility in the index and middle fingers produced by the nerve injury (*vide* fig. 3, H, p. 136).

In Case 13 (Table I., p. 152) we had the opportunity of measuring the growth of the nails after the median nerve had been divided without injury to the tendons, and found that they grew equally on the two sides. Then the wound was explored and the two completely separated ends were freshened and reunited.

For sixteen days the hand remained in splints and the nails grew to the following amount:—

	Right (*affected*).	*Left* (*sound*).
Thumb	1 mm.	3 mm.
Index	2·5 mm.	3 mm.
Middle	1·5 mm.	3·5 mm.
Ring	2·5 mm.	3 mm.
Little	2 mm.	3·5 mm.

But as soon as the hand was freed, the nails again grew equally, in spite of the total cutaneous insensibility of the index and middle fingers.

Complete loss of sensibility to light touch and to the minor degrees of heat and cold makes no difference to the growth of the nails. In Case 12 (Table I., p. 152, and fig. 3, F, p. 136) all sensation to light touch was lost over the full median area and yet for sixty-three days the nails grew equally on both hands. During this period all movements of the hand were perfect except those of the abductor and opponens pollicis.

Sometimes an incomplete division of the median produces the full loss of sensation that usually follows complete division of the nerve, without muscular paralysis. An instance of this condition is to be found in Case 29.[1] This youth showed to a profound degree the influence of immobilisation. During the thirty-two days after the operation when the hand remained on a splint the nails grew very little on the affected side.

	Left (affected).	*Right (sound).*
Thumb	1·5 mm.	4 mm.
Index	0 mm.	2 mm.
Middle	0 mm.	2·5 mm.
Ring	1·5 mm.	3·5 mm.
Little	2 mm.	3 mm.

But during the following thirty-five days the nails on the two hands grew equally in spite of the loss of sensation.

Loss of sensation alone, whether complete or partial, makes no material difference to the growth of the nails. How, then, are we to explain the profound alteration produced by division of the ulnar nerve?

A characteristic instance of this defective growth was seen in Case 63, where sensation was lost over the full ulnar area (fig. 1, H, p. 127). No tendons were divided and the following differences must have been due solely to the consequences of division of the ulnar nerve. In one hundred and thirty days, during which sensation showed no sign of return, the growth was as follows:—

	Right (affected).	*Left (sound).*
Index	16 mm.	16 mm.
Middle	15 mm.	17 mm.
Ring	11·5 mm.	14·5 mm.
Little	10 mm.	14 mm.

But this result, so characteristic of lesions of the ulnar nerve, cannot be due to any direct effect of the nerves upon the growth of the nails; for in a woman, aged 36 (Case 23, Table II., p. 158), in whom the ulnar nerve had been reunited, the change was equally definite, although all sensation had returned. But at this time none of the muscles of the hand supplied by that nerve were acting voluntarily.

---

[1] Reported in full in the Appendix, p. 302, *vide* also Table IV., p. 164.

	Right (*affected*).	*Left* (*sound*).
Index	7 mm.	8 mm.
Middle	6 mm.	8 mm.
Ring	5 mm.	9 mm.
Little	4 mm.	7 mm.

Here the paralysis alone must have been the cause of this difference in growth, a difference which can be partly prevented by the use of regular massage. Case 19,[1] shows how closely the deficient growth after division of the ulnar nerve depends upon want of movement. At first, during the thirty-four days the hand was on splints, the nails grew to the following extent:—

	Left (*affected*).	*Right*.
Thumb	1·5 mm.	4 mm.
Index	1 mm.	—
Middle	2·5 mm.	4 mm.
Ring	2·5 mm.	4 mm.
Little	2 mm.	4 mm.

During a period of forty-nine days, when all sensation was lost over the ulnar area, their growth was as follows:—

	Left (*affected*).	*Right*.
Thumb	6 mm.	7 mm.
Index	6·5 mm.	7 mm.
Middle	6·5 mm.	6·5 mm.
Ring	6·5 mm.	6 mm.
Little	5 mm.	6 mm.

But during this time the hand was massaged regularly three times a week. Later this treatment was discontinued, and for forty-nine days he relapsed into the deficient growth so characteristic after division of the ulnar nerve.

---

[1] Reported in full in the Appendix, p. 309.

	Left (affected).	*Right.*
Thumb	6 mm.	5 mm.
Index	4 mm.	5 mm.
Middle	4 mm.	6 mm.
Ring	3 mm.	6 mm.
Little	1·5 mm.	4·5 mm.

The fact that the nails of the middle, ring and little fingers, and not infrequently that of the index, are affected after injury to the ulnar nerve is sufficient alone to show that its sensory branches can have little to do with this characteristic change.

In conclusion, we believe that the most potent cause of diminished growth in the nails after division of a peripheral nerve is want of movement. Whenever the skin becomes insensitive and the injury has not divided tendons or paralysed muscles, the nails do not show any deficiency in growth. The profound alteration that follows division of the ulnar nerve is produced by paralysis of the intrinsic muscles of the hand, and stands in no relation to the loss of sensibility.

When the skin of the hand becomes glossy and when hyperalgesia is well developed, the nails undergo a change radically different from that seen after division of a nerve. Weir-Mitchell says: "When the depraved nutritive state (glossy skin) has lasted for some months, the hair commonly disappears from the fingers affected, and the nails undergo remarkable alterations. . . . The alteration in the nail consists of a curve in its long axis, an extreme lateral arching, and sometimes a thickening of the cutis beneath its extremity. In other cases a change takes place which is quite peculiar, or which to us at least was new. The skin at that end of the nail next to the third finger joint becomes retracted, leaving the sensitive matrix partly exposed. At the same time the upper line of union of skin and nail retreats into or under the latter part, and in place of a smooth edge is seen through the nail as a ragged and notched border."

In the only instance of true glossy skin that has come under our notice (Case 63) the nails of the little and ring and middle fingers curved longitudinally and horizontally.

They were not ribbed but were thin and exquisitely tender. The patient was certain that these nails grew faster than those on the sound hand; but owing to the shortness of the period during which he was under our observation before the nerve was divided and sutured, we were unable to verify his statement by measurement. The remaining instance of hyperalgesia did not affect the tips of the fingers, and threw no light on this increased growth of the nails. But whenever a curved nail is growing slowly it is thick and hard. In the case of L. G. H. (No. 63) the nails were, on the contrary, smooth and thin, collateral evidence that they were growing faster than normal.

CHAPTER 15.—PARALYSIS AND OTHER MUSCULAR CHANGES.

The motor supply of one nerve rarely overlaps that of another, and can be readily determined by dissection. The examination of cases where a peripheral nerve has been divided has not led us to doubt the usual teaching concerning the supply of any of those muscles of the limbs that have come under our observation. This chapter will therefore be devoted mainly to a consideration of the time required for the recovery of muscular power, the disappearance of the wasting and the restoration of irritability to the interrupted current.

Before passing to the results of our observations, we wish to call attention to some possible sources of error in the methods usually employed to determine whether a muscle is acting voluntarily.

The little finger is abducted by the combined action of two muscles, the abductor and the extensor minimi digiti. If the former is paralysed, as is the case after division of the ulnar nerve, false abduction can be produced by means of the extensor. The nature of this movement can be at once recognised by the extension which accompanies it. When the hand is placed flat on the table the little finger is seen to be raised if the abduction is caused by the extensor only.

Another fruitful source of error, as pointed out by Beevor (1), is false abduction of the thumb by means of its extensor muscles. True abduction of the thumb takes place in a plane at right angles to that of the palm, and this is the action of the abductor pollicis. But under ordinary circumstances this muscle acts in combination with the extensors, and when the abductor and opponens pollicis are paralysed from division of the median nerve, the extensor muscles alone can produce some abduction. The true nature of this movement is betrayed by the impossibility of performing it without extending the thumb.

In like manner, the movement produced by the opponens can be simulated by contraction of the flexor longus pollicis and of the adductors of the thumb. The true mechanism of this movement can be recognised by the flexion of the terminal phalanx with which it is associated.

The index finger can be abducted slightly by means of its extensor, and this may cause some difficulty, after division of the ulnar nerve, in determining whether the first dorsal interosseous is acting.

Another difficulty in connection with this muscle arises when it is tested electrically. After the ulnar nerve has been divided the first dorsal interosseous muscle wastes. But in the position of the wasted muscle, contraction can be frequently obtained by means of the interrupted current. This is due to stimulation of the first lumbricalis inserted into the dorsal expansion of the extensor tendon and supplied by the median nerve.

Division of a motor nerve causes immediate paralysis in the muscles it supplies. But they continue to react to the interrupted current for from three to five days. After the fourth to the seventh day we obtained no response to the interrupted current in the muscles of the hand supplied by the median or by the ulnar nerve. Here our experience coincides with that of Bowlby (2), who states that he has been unable to obtain any reaction to the strongest current as early as the third or fourth day after division of the nerve. Statements assigning a considerably later date are probably vitiated by the inclusion of cases of injury or incomplete divided, the muscles first responded to the interrupted current shortly before any voluntary contraction could be obtained. One case only showed any return of voluntary power before a reaction was obtained to the interrupted current.

Voluntary power ultimately returned to all the paralysed muscles in every patient whom we have observed from the time when the nerves were sutured.

In order to discover if, in some instances, the paralysed muscles did not recover, we examined a number of patients in whom primary suture of one or more of the nerves to the hand had been performed at the London Hospital before the beginning of our research. Of four cases where the ulnar had been divided, one showed complete paralysis of all the muscles supplied by that nerve five and a half years after its suture. All the others had recovered when first seen by us from two to four years after the injury.

Five of the six patients in whom the median nerve had been divided had completely recovered when we first saw them two to four and a half years after suture; one still showed no return of motor power eighteen months after the nerve had been reunited.

Thus, we may say that most patients regain voluntary power in the affected muscles after primary suture of one or more of the nerves of the hand; but the strength of the contraction and the ability of the hand not infrequently remain perman [illegible]

Primary su [illegible] the ends of a nerve within [illegible] variable in the case is [illegible] turn of accidental wound has healed, and cases of secondary suture are rare. We have as yet been able to follow six patients only up to complete recovery of voluntary power and muscular reaction. Among them the time at which the ends of the nerve were reunited varied from 15 to 502 days after division.

The following table shows these cases and the time of their recovery :—

Ulnar Nerve.

	Period after Division at which Secondary Suture was Performed.	Return of Voluntary Power.	Return of Reaction to the Interrupted Current.
No. 18	15 days	702 days	702 days.
,, 23	28 ,,	408 ,,	408 ,,
,, 24*	502 ,,	370 ,,	370 ,,

Median Nerve.

No. 85*	66 days	816 days	635 days.
,, 12	153 ,,	481 ,,	369 ,,
,, 11	375 ,,	373 ,,	373 ,,

It will be noticed that there is no instance among them of that remarkable recovery of muscular power recorded by Kennedy (7) after secondary suture. The earliest return of voluntary power occurred in 370 days after the nerves had been reunited.

We possess among our records one complete case of secondary suture of the musculo-spiral nerve (No. 43, reported in full in t[illegible]ry power reappeared i[illegible]after the ends of the[illegible]was intolerant complete section. Whenever the nerve is injured severely, recovery takes place extremely slowly; in the only instance where it was completely divided voluntary power had not returned to the paralysed muscles three and a half years after the ends of the nerve had been reunited.

When a nerve is incompletely divided the injury may produce the most varying results upon muscular power and on the reaction to electrical stimulation. In many cases voluntary power may not be lost in the muscles supplied by the injured nerve, and they may react normally to both forms of electrical stimulation. Voluntary power may remain, but reaction to the interrupted current be lost; the muscles will then respond more readily to the constant current. The contraction still occurs to the negative pole more readily than to the positive, but the strength of current necessary to cause contraction is considerably reduced. This facile reaction to the constant current also occurs after incomplete division of the nerve when voluntary power is lost, and all response to the interrupted current is abolished. It is therefore a valuable indication that the nerve has not been completely severed.

CHAPTER 16.—THEORETICAL.

The observations detailed in the previous chapters are so compl [illegible] view of the mechanism of sen [illegible] it will be well to summa [illegible] o-ordinate them into a [illegible] over the affected parts. When the median nerve was divided, stimulation with the feathers of a quill or with the head of a pin could be appreciated in some cases over the whole area supplied by that nerve, including even the fingers. But these parts were entirely insensitive to temperature, and the compass points could not be discriminated even when 6 cm. apart.

He showed that, after division of the median nerve, the movement resembling opposition of the thumb was due to contraction of its adductor and flexor muscles. He also recognised false abduction, attributing it to "l'action du long abducteur du pouce" (extensor ossis metacarpi pollicis).

On the sensory side, he believed the absence of complete insensibility was due to anastomosing branches and to the conduction of mechanical vibrations on to neighbouring parts where the nerve end organs were intact.

His observations were correct and his criticisms just. But unfortunately surgeons failed to appreciate the significance of this work, although ready to invoke "supplementary sensation," when the condition of sensibility did not accord with their expectations. Thirty years after the publication of Létiévant's book, neglect of his warning has led to the advent of another generation of robust believers, who report cases where suture of divided nerves has led to immediate or strikingly rapid return of sensation.

Létiévant and hi [illegible] thing of afferent fibres [illegible] o them the conception [illegible] us was therefore im [illegible]

Complete division of all the sensory nerves to the skin leaves the part sensitive to those stimuli commonly employed by the surgeon as a test for sensibility to touch.

All forms of pressure, such as a touch with a pencil or the feathers of a quill, can be appreciated and localised with considerable accuracy. Two points applied successively can be recognised, but, if applied simultaneously, the patient entirely fails to discriminate them, even when the compasses are widely separated. The denervated part is insensitive to all forms of heat and cold. Pain can only be evoked by pressure, and then has that peculiar aching character associated with a crush or contusion.

We have shown that this deep sensibility is restored rapidly, and seems to reach the hand by way of the tendons and fibrous structures connected with them. Division of the median nerve without injury to these structures leaves the response to all forms of pressure almost unaffected.

Clear recognition of these facts is necessary before we can attempt to explain the condition of cutaneous sensibility after division of a peripheral nerve. Stimulation can rarely be made without some pressure, but every stimulus must be of so specific a nature that the pressure element in the sensation passes into the background. A pin-prick may be appreciated and localised, but, unless it is recognised as causing pain, its appreciation may have been entirely due to deep s … pri … ck with a pin was felt an … of the presence of

uta

as the median

and 40° C., although within its borders ice and water at 50° C. can be appreciated.

Moreover, if a nerve has been completely severed, recovery of sensation does not take place, as is usually believed, by a gradual increase in sensibility, beginning in parts where sensation has never been lost entirely; but the hand first becomes sensitive to prick and to the more extreme forms of heat and cold. Only after an interval of some months do the higher forms of sensibility begin to return.

The intermediate zone and a hand in the first stage of recovery are alike in their insensibility to cotton-wool and to temperatures between about 25° C. and 40° C.; the compass test fails utterly, even when the points are separated to many times the distance necessary upon the normal hand. All the finer and more delicate sensations involving discrimination and differentiation are wanting. Alike in these defects, the sensibility of the intermediate zone and that of the recovering hand resemble one another in the peculiar character of their response to stimulation. A prick causes immediate withdrawal of the hand with evident signs of discomfort, and the sensation it produces is badly localised, radiating widely over the parts affected. Stimulation with temperatures below 20° C. evokes a sensation of cold which radiates widely and is of a tingling character. If the point of a pin is dragged lightly across the skin from normal to abnormal parts, sensa [illegible] the line is crossed at which li [illegible] There is no gradual pas [illegible] of abnormal sensibil [illegible] greater than that from the normal skin. A prick causes instant withdrawal of the hand, with evident signs of extreme discomfort. Ice and water at 50° C. seem respectively colder and hotter over the affected area than over normal parts of the hand. Sensation is less acute, but more vivid than that from the normal skin.

Thus the sensibility of the intermediate zone and that of the recovering hand are similar in kind; but, whilst the latter reacts more briskly than normal to its peculiar stimuli, sensation over the intermediate zone is obviously defective. The intermediate zone, apart altogether from its insensitiveness to light touch, may be rightly spoken of as an area of diminished sensibility.

This form of sensibility, so characteristic of parts to which sensation is returning, we call *protopathic.*

It will be well to formulate the essential characteristics of this form of sensibility as manifested in a hand that has reached the end of the first stage of recovery. Every stimulus, to which the part reacts, produces a sensation that radiates widely and is accompanied by a peculiar tingling quality. The point of stimulation is recognised with considerable accuracy in consequence of the sensibility of the subcutaneous structures to the pressure that necessarily accompanies almost every stimulus. But the specific sensation of pain, of cold, or of heat, seems to be situated in some remote part, such as the fingers, or to extend widely over the palm. Sometimes an intelligent patient will say: "You touched me on the palm, but the prick is all over the fingers." As far as we have observed, the radiation never spreads to parts over which light touch is perfect and the compass test gives a normal record; conversely, a prick over normal parts does not produce any sensation within a contiguous area of protopathic sensibility.

Although parts in this condition react more vehemently to painful stimulation than those that are unaffected, the stimulus necessary to evoke sensation appears to be greater. When the normal hand is pricked with a pin, a sensation of sharpness is produced almost as soon as the point touches the skin. Over protopathic parts the point must be applied more firmly before pain is produced, and this sensation not only arises more slowly, but lasts after the stimulus is removed. The widespread, aching pain produced by a prick of just sufficient force to arouse the sensibility of a protopathic area is more intolerable than the sensation caused by a prick of considerable violence over normal parts. The patient cries out, and withdraws his hand rapidly. Thus, although a stronger stimulus is necessary to evoke a painful response from parts in a condition of protopathic sensibility than from those where sensation is normal, the discomfort manifested by the patient is obviously greater.

Temperatures below 40° C. do not, as a rule, evoke a sensation of heat from parts in this condition, when the patient belongs to the ordinary hospital class. But, in one of us, the back of the hand, endowed only with protopathic sensibility, habitually reacted to 38° C. over one small area of maximal sensibility. Over parts at the end of the first stage of recovery, water at 45° C. will certainly be recognised as warm during the summer months; but over the intermediate zone with its lowered protopathic sensibility, or when the cold of winter has rendered the affected parts less sensitive, a temperature of 50° C. may be required before any sensation of heat is produced. Such a temperature is capable of producing pain, even over the normal hand, and it might be objected, that the sensation it produces over such protopathic parts is in reality one of pain rather than of temperature. But the patient states, that though pain and tingling are evoked by stimuli at this temperature, they are accompanied by a definite sensation of heat.

An area supplied only with protopathic sensibility reacts more vehemently than normal parts to all temperatures capable of evoking a response. If a test tube containing water at 45° C. is moved across the hand from normal to abnormal parts, it appears ~~to become~~ hotter as soon as the protopathic area is reached. And yet this same area is totally insensitive to water at 35° C., which is decidedly warm to the normal hand. Thus, the increased reaction to 45° C. over the protopathic area is in no way due to an increased sensitiveness to heat as a whole.

dragged across the skin. If, when the hand is in this condition, a test tube containing water at 45° C. is passed across the palm, it appears to grow hotter over the protopathic area. But when the same procedure is carried out with a test tube at 37° C. it appears to grow cooler as soon as the affected parts are reached. The recovering parts in this experiment react less briskly to the one temperature (37° C.) and more briskly to another (45° C.) than parts of normal sensibility.

To cold stimuli, especially when the temperature is below 18° C., the reaction of protopathic parts is equally characteristic; no stimulus produces more striking radiation and diffusion. The sensation of cold appears to extend widely, or to be situated in some distant part of the affected area. Moreover, the reaction produced by temperatures below 18° C. is greater over protopathic than over normal parts; the stimulus seems to the patient to be colder.

If the affected part happens to be endowed with hair, it will be found that many of the hairs when pulled cause pain. This pain is not localised, but radiates widely. But, in addition, any movement, of these hairs, such as is produced by brushing the part lightly with cotton-wool, will cause a sensation differing from that over normal parts. For not only does it consist of a curious tingling, or formication, but it radiates widely and is frequently referred to parts at a distance. This sensation can be evoked only by stimulating the hairs, and, unlike the normal skin, the part becomes entirely insensitive to cotton-wool when shaved.

Whatever may be the stimulus that evokes a sensation from protopathic parts, that sensation is always characterised by a "tingling" quality and by defective localisation. So erroneous may be this localisation, that although the impact of the stimulating body is perceived and localised correctly recovery of sensation, but would have no further significance.

But everything seems to point to the introduction of a new factor rather than to the gradual improvement of a function already present. For, before the advent of light touch, the patient could appreciate correctly the point which had been pricked, or the area stimulated with ice, by means of the pressure so produced. But this did not hinder wide radiation of the specific sensation. This radiation is only brought to an end by the return of sensibility to light touch and the recovery of power to discriminate two compass points.

All power of localisation present before the return of light touch must have been due to what we have called deep sensibility. It is conceivable that the final disappearance of radiation and the other protopathic characteristics might be due to the development of a new quality that made localisation in the skin a possibility. This quality would be in some way associated with the return of sensibility to light touch. But this return could have no direct effect upon sensations of temperature, and yet one of the most definite features of this stage of recovery is the appreciation of intermediate degrees of temperature to which the part in the protopathic condition was insensitive.

Moreover, the return of sensibility to light touch seems so closely bound up with the recovery of sensation to intertheir reappearance within the affected area is accompanied by a coincidental decrease in radiation.

The use of these terms, "protopathic" and "epicritic," would be convenient even if they represented nothing but stages in recovery after division of a peripheral nerve. But we believe that each corresponds to the function of a distinct system of nerve fibres and end organs.

In a previous part of this paper (Chapter 1) we showed from a series of cases that the area rendered insensitive to light touch by division of the median or of the ulnar nerve varied little in extent.

In sharp contrast to this slight variation stood the extreme differences in extent of the loss of sensation to prick which followed division of either of these nerves. So greatly did the area of cutaneous analgesia vary in each individual instance that it was impossible to formulate any general statement concerning the normal extent of the area rendered insensitive to prick by division of the median or of the ulnar nerves. Moreover, the extent of the loss of sensation to light touch and to prick vary independently of one another. The most extensive cutaneous analgesia is not necessarily associated with an increased area of insensibility to light touch. Conversely, when the loss of sensation to prick occupies but a small extent of the hand, or is confined to the fingers, the area insensitive to light touch is not of necessity smaller in proportion. This want of relation between the extent of the loss of sensation to prick and to light touch after complete division of a nerve renders it unlikely that the two forms of sensibility are due to the same anatomical system of nerve fibres and end organs.

When the ends of a divided nerve have been successfully sutured, protopathic sensibility not only returns first, but the whole of the affected parts may remain for many months entirely insensitive to all the higher stimuli. If protopathic and epicritic sensibility were only functional modifications in the activity of one anatomical system, it would be difficult to explain how the complete restoration of the one could leave entirely unaffected the extent of the area over which the other was absent. The improvement should be general, and should lead to a gradual retreat of the borders of the area insensitive to the higher stimuli step by step with the disappearance of the cutaneous analgesia. This actually occurs when a nerve has been injured and not completely divided. After such a lesion no widespread regeneration is necessary; the nerve fibres have but to recover their function temporarily in abeyance. But, after suture of a completely divided nerve, the two systems of fibres evidently regenerate with unequal facility, and thus the one form of sensibility is re-established before the other shows any signs of return. The results that follow the unaccompanied restoration of protopathic sensation have been fully described when we considered the condition of a part at the end of the first stage of recovery.

Protopathic sensibility is restored under conditions which materially hamper the return of the higher forms of sensibility. The formation of fibrous tissue between the two ends of a nerve greatly retards the restoration of sensation, but, when this fibrous tissue is removed at the operation for secondary suture, it sometimes happens that the extent of the protopathic loss is increased (Case 11, Case 28). This increase must have been due to the removal of nerve fibres intermingled with the fibrous tissue, which were capable of endowing the part with protopathic sensibility. Here a condition capable of preventing the return of the higher forms of sensation did not form an effectual bar to the regeneration of the fibres subserving protopathic sensibility.

With two systems of nerve fibres we should expect that occasionally injury of a peripheral nerve would produce the converse form of dissociated sensibility. On this hypothesis it is unlikely that the fibres would be distributed uniformly to every peripheral nerve, and we should find occasionally that a part insensitive to prick reacted to the lightest touch. Such a dissociation is rare, but seems to exist especially tested with the compasses.

The opportunity of testing an area of so considerable a size seldom arises, but in the case of one of us, after division of two nerves in the forearm, a triangular area insensitive to prick, but sensitive to light touch, made its appearance in the neighbourhood of the wrist. Here ice and water at 50° C. were not appreciated, but sensation seemed to be retained to temperatures between 36° C. and 45° C.

In Case 85, a small patch of dissociated sensation appeared on the back of the hand in consequence of division of the median and part of the radial and external cutaneous nerves at the wrist. Here the patient could appreciate stimulation with cotton-wool and with warmth, but was insensitive to prick and to the application of ice.

Thus, in conclusion, we believe that the following reasons render it probable that what we have called "protopathic" and "epicritic" sensibility depend on two anatomically separate systems of fibres and end organs.

Firstly, it is difficult to see how else can be explained the want of relation between the extent of the area rendered insensitive to light touch and that insensitive to prick after division of a peripheral nerve. Secondly, the comparatively early return of protopathic sensibility after suture of a completely divided nerve brings with it no diminution in the area insensitive to light touch and minor degrees of temperature. Whereas, if the nerve has been injured, but not completely divided, the two forms of sensibility return step by step. Thirdly, we have found on rare occasions that, after division of a peripheral nerve, a small portion of the insensitive area may react to stimulation with cotton-wool and to minor degrees of heat, but not to prick or to ice.

We have now reached the conclusion that every part of the limbs and surface of the body possesses three systems of afferent fibres. The first of these runs with the motor nerves, and is not destroyed by the division of all cutaneous sensory branches. These afferent fibres supply the part with deep sensibility, and are responsible for much of the sensation that remains after division of peripheral nerves.

They run with the motor nerves from the periphery to the point where motor and sensory fibres separate, forming the anterior and posterior roots. Here they join the posterior roots and pass into the posterior columns of the spinal cord. In the case of the hand, we brought forward evidence to show that deep sensibility was materially diminished by division of the long tendons at the wrist. The afferent fibres upon which this sensibility depends must therefore have passed from the palm and fingers along the tendons; they then join in the forearm, the motor fibres to that muscle of which the tendon is only a prolongation. If we know the anterior root by which these motor fibres pass to innervate the muscle, we may assume that the equivalent posterior root will carry the afferent fibres connected with the tendon and its fibrous projection. Given the tendons and the aponeuroses which are necessary for the maintenance of deep sensibility to any part, we can then work out its segmental innervation.[1]

The observations detailed in this paper do not permit us to make any more definite statement with regard to the distribution of these afferent fibres concerned with deep sensibility. The question of the nature and extent of pain arising in connection with this system must be reserved for a future communication.

The laws which govern the two cutaneous systems can be laid down with greater certainty, at any rate as far as the limbs are concerned. In many ways the supply of epicritic sensibility is the simpler, especially from the peripheral aspect. We shall therefore begin with the epicritic supply of

[1] We have spoken throughout as if no fibres existed in the skin concerned with the conduction of pressure impulses. It must not be supposed that we do not believe such fibres exist. We are compelled to neglect them because these fibres are probably removed when all cutaneous sensory nerves have been divided. Since our only knowledge of the properties and distribution of deep sensibility can be gained from parts that have been deprived of all their cutaneous sensory nerves, any fibres concerned with deep sensibility existing in the skin are outside the limits of our method of observation.

the arm and hand; then we shall consider their protopathic innervation, and, finally, we shall apply the laws so established to the cutaneous nerves of the leg.

We have shown that the epicritic supply of the median and ulnar nerves overlaps little on the palm. When the ulnar is divided, the residual sensibility, maintained by the median, does not extend further on the palm than a line drawn through the axis of the ring finger; nor does it occupy more than two-thirds of that finger. In no instance has the median been capable of supplying epicritic sensibility to the whole of the ring finger after complete division of the ulnar nerve. Conversely, after division of the median, the radial half of the palm has in every case been insensitive up to a line drawn from the cleft between the middle and ring fingers. This boundary has a sinuous outline, but, roughly speaking, it corresponds to the vertical line just described. In no instance has the intact ulnar been able to innervate the whole of the ring finger after division of the median. These two nerves, as far as their epicritic supply is concerned, cannot overlap one another by the breadth of a single finger.

In the same way the border on the thenar eminence between the supply of the median and that of the combined pre-axial group of nerves (ramus cutaneus n. radialis and external cutaneous) is singularly constant and definite. After the median has been completely divided, the borders of the loss of sensation on the thumb and outer part of the thenar eminence correspond almost exactly to the radial boundary of the area that becomes insensitive to epicritic stimulation after destruction of the pre-axial group of nerves in the forearm.

Whenever the adjacent borders of two anæsthetic areas correspond closely in this manner, the two nerves by which they are supplied can overlap little as far as that particular form of sensibility is concerned.

On the back of the index and middle fingers the epicritic supply of the median, overlaps that of the pre-axial group by rather less than three-fourths of the extent of the basal phalanx.

method of residual sensibility the amount of overlapping between the posterior roots that innervate the hand, he wished to see to what extent this overlapping was represented in the median and ulnar. "It is then clear," he says, "that in the hand . . . . of Macaeus the extent of overlap of the skin fields of the peripheral nerve trunks, even on the exquisitely sensitive . . . . palmar surface is much less than that of the cutaneous areas of the nerve roots; it is, in fact, not so great as may be the overlap of the fields of nerve roots three segments distant from one another."[1]

This complete accord between the results of our observations on man, and those made by Sherrington on monkeys, shows that the stimuli he used appealed to what we have called the epicritic system.

When we turn to the distribution of protopathic sensibility we come face to face with an arrangement fundamentally different. Division of the median or the ulnar nerve produces loss of protopathic sensibility over a comparatively small area with indefinite borders. The residual sensibility to protopathic stimuli present after the median nerve has been divided extends, in many instances, over the whole palm. After division of the ulnar the palm remains sensitive to such stimuli everywhere, except over the extreme ulnar border. Moreover, when the trunk of the ulnar nerve was irritated (Case 63, p. 316), the tenderness spread across the palm to the thumb and base of the index and middle fingers. The consequences of both division and irritation of these nerves show that, as far as protopathic sensibility is concerned, they overlap to an enormous extent.

The anatomical expression of this overlapping is found in the anastomotic branches which probably exist in order that the fibres subserving protopathic sensibility may pass from the territory of one nerve into that of the other.

On the back of the hand the ulnar and internal cutaneous extend as far as a line that corresponds roughly with the tendon of the middle finger. This post-axial group also supplies protopathic sensibility to the whole of the ring finger, except about one-third of the radial aspect of the terminal two phalanges; it also sends fibres to the ulnar half of the basal phalanx of the middle finger. The careful and laborious dissections of Brooks (3) and Hédon (6) bring out these points admirably, and show that the branches traced by them across the usual anatomical borders subserve the overlapping protopathic sensibility.

---

[1] *Phil. Trans. Roy. Soc.*, 1898, vol. cxc., p. 109. Compare also the figures on p. 108, which are wrongly numbered; the figure on the left of the reader is No. 2, that on the right No. 1.

On the forearm division of the radial (ramus superficialis n. radialis) and external cutaneous produces an area of protopathic insensibility with a well-defined border on the flexor surface of the forearm. This border corresponds closely, except at the wrist, with that of epicritic loss; but at the wrist the protopathic fibres of the post- and pre-axial groups overlap greatly, and the loss of sensation to prick is less extensive than that to light touch.

Everywhere in the forearm and hand division of a peripheral nerve causes loss of protopathic sensation over an area of smaller extent than the accompanying epicritic insensibility; but, as soon as the nerve is injured on the central side of the brachial plexus, and especially if it be the roots that have suffered division, this rule may be reversed. The loss of protopathic sensibility exceeds in extent the area insensitive to epicritic stimuli, and the skin may be in parts sensitive to light touch, but not to prick (*vide* Case 52, p. 194).

The nearer the lesion is situated to the posterior roots the more extensive and definite is the loss of protopathic sensibility; the more nearly the injury divides one of the nerve groups described above, such as the median, the ulnar, or the pre-axial nerves, the more definite and extensive is the epicritic loss. It would seem, then, as if each of these peripheral nerves, or nerve groups, formed a unit of the epicritic system; the protopathic unit must be sought in one or more posterior nerve roots.

This does not imply that division of a single posterior root would produce an area insensitive to protopathic stimuli, but sensitive to light touch and to the minor degrees of heat and cold. For even from the protopathic aspect, fibres of any two posterior roots overlap one another especially on the limbs. But this overlapping is considerably less for the protopathic fibres of any one posterior root than for those which subserve epicritic sensibility.

From this it follows that the area which becomes totally insensitive to protopathic stimulation after a peripheral nerve has been divided, represents the extent of skin supplied by those fibres of one or more posterior roots which run exclusively in that nerve. For instance, the area of total cutaneous insensibility which follows division of the ulnar, represents that part of the hand supplied solely by those protopathic fibres of the first dorsal root, which run in the trunk of that nerve. Every other part of the hand supplied by fibres that run in the first dorsal posterior root, will remain sensitive to protopathic stimuli because it is innervated also from the median.

If, then, it should happen that the area supplied by a peripheral nerve coincided with that of one or more posterior roots, division of that nerve would produce a patch of total cutaneous insensibility co-terminous for both epicritic and protopathic stimulation.

The external popliteal, including its lateral cutaneous branch, closely corresponds to such a nerve, excepting in the region of the knee and outer side of the foot. Complete division of this nerve produces an area of epicritic loss, bounded by a line which slants across the shin to a point just in front of the inner malleolus. In the region of the calf the boundary of the loss of this form of sensation runs vertically down the leg to the outer side of the tendo-Achillis.

Both these lines also form the boundaries of the loss of protopathic sensibility, so that here we find a remarkable coincidence of the borders of the loss to protopathic and epicritic stimuli, a condition foreshadowed by the consequences that follow division of the internal cutaneous in the forearm.

The relation of the other nerve branches in the leg is complex, and furnishes no clear example of the laws we have here laid down. The detailed discussion of their distribution will be found in Chapter 7.

these systems has one set of end organs only, and that each end organ is sensitive to every form of stimulus to which that system responds. In a subsequent paper it will be shown that, as far as protopathic sensibility is concerned, at least three end organs exist and each of these reacts only to a specific stimulus. In the present communication we are dealing only with the distribution of the fibres that underlie the two main forms of cutaneous sensibility.

In conclusion, we believe that the afferent fibres in the peripheral nerves can be divided into three systems.

(1) Those which subserve deep sensibility and conduct the impulses produced by pressure. The fibres of this system run mainly with the motor nerves, and are not destroyed by division of all the sensory nerves to the skin.

(2) Those which subserve protopathic sensibility. This system of fibres and end organs responds to painful cutaneous stimuli, and to the extremes of heat and cold. It also endows the hairs with the power of reacting to painful stimulation.

These fibres regenerate rapidly after the ends of the nerve have been reunited; if the operation has been successfully performed sensation begins to return within from seven to ten weeks.

In any peripheral nerve the distribution of the protopathic fibres usually overlaps greatly the area supplied by the fibres of the adjacent nerves.

(3) Those which subserve epicritic sensibility. The nerve fibres and end organs of this system endow the part with the power of responding to light touch with a well-localised sensation. The existence of this system enables us to discriminate two points and to appreciate the finer grades of temperature called cool and warm.

These fibres regenerate more slowly than those which subserve protopathic sensibility after reunion of a divided nerve, and sensation does not usually begin to return in less than six months under the most favourable conditions.

The distribution of these fibres in the larger peripheral

Until February 4, the hand and fingers were kept at rest and the nails grew unequally.

	Left (affected).	*Right.*
Thumb	0	·5·5 mm.
Index	0	5·5 mm.
Middle	0	5·5 mm.
Ring	0	5 mm.
Little	0	5·5 mm.

He started work on June 3, and used both hands alike; the nails now grew to an equal extent on both hands.

	Left (affected).	*Right.*
Thumb	7 mm.	6 mm.
Index	6 mm.	6 mm.
Middle	6 mm.	6 mm.
Ring	6 mm.	6 mm.
Little	6 mm.	6 mm.

By September 6, he could appreciate light touch and water at 22° C. and at 40° C. over the proximal portion of the affected half of the palm; over these parts the compass test was good at 2 cm.

On September 30, the opponens and abductor muscles acted voluntarily and reacted to the interrupted current. Cotton-wool and the minor degrees of temperature were appreciated over the whole affected palm on this date, but the fingers remained anæsthetic until November 18.

A marked line of change to prick was present, bounding the old anæsthetic area until June, 1905. By August 21, 1905, this had disappeared, and the compass test was perfect at 1 cm.

Case 6.—Division of the median nerve. Recovery thrown back by the formation of an abscess at the site of the healed wound. Trophic disturbances of the skin.

S. H., a barber, aged 21, cut his right wrist with a razor on October 3, 1903. The wound was explored the following day; the median nerve, the tendons of the flexor sublimis digitorum, flexor longus pollicis, and several of those of the flexor profundus, were found to have been divided. The nerve was sutured with silk, the tendons with catgut.

On January 13, 1904, when he first came under our observation, the outer group of thenar muscles was markedly wasted, but contracted voluntarily and reacted to a strong interrupted current.

Until February 4, the hand and fingers were
the nails grew unequally.

	Left (affected).	Right.
Thumb	0	·5·5 mm.
Index	0	5·5 mm.
Middle	0	5·5 mm.
Ring	0	5 mm.
Little	0	5·5 mm.

He started work on June 3, and used both hands alike; the nails now grew to an equal extent on both hands.

	Left (affected).	Right.
Thumb	7 mm.	6 mm.
Index	6 mm.	6 mm.
Middle	6 mm.	6 mm.
Ring	6 mm.	6 mm.
Little	6 mm.	6 mm.

By September 6, he could appreciate light touch and water at 22° C. and at 40° C. over the proximal portion of the affected half of the palm; over these parts the compass test was good at 2 cm.

On September 30, the opponens and abductor muscles acted voluntarily and reacted to the interrupted current. Cotton-wool and the minor degrees of temperature were appreciated over the whole affected palm on this date, but the fingers remained anæsthetic until November 18.

A marked line of change to prick was present, bounding the old anæsthetic area until June, 1905. By August 21, 1905, this had disappeared, and the compass test was perfect at 1 cm.

Case 6.—Division of the median nerve. Recovery thrown back by the formation of an abscess at the site of the healed wound. Trophic disturbances of the skin.

S. H., a barber, aged 21, cut his right wrist with a razor on October 3, 1903. The wound was explored the following day; the median nerve, the tendons of the flexor sublimis digitorum, flexor longus pollicis, and several of those of the flexor profundus, were found to have been divided. The nerve was sutured with silk, the tendons with catgut.

On January 13, 1904, when he first came under our observation, the outer group of thenar muscles was markedly wasted, but contracted voluntarily and reacted to a strong interrupted current.

The skin of the median half of the affected palm was dry and harsh, contrasting in a marked manner with the smooth, moist appearance of the unaffected portion. On the radial side of the tips of the index and middle fingers were blisters ; these he had noticed on waking three mornings previously. They had not been present when he went to bed, and were caused by no known injury.

He was insensitive to stimulation with cotton-wool, and the minor degrees of temperature over the area shown in fig. 31. Over the terminal two phalanges of the index and middle fingers, the palmar surface of the terminal phalanx of the thumb, and the extreme radial border of the last two phalanges of the ring finger, he failed to appreciate a prick, ice, water at 60° C., and pressure.

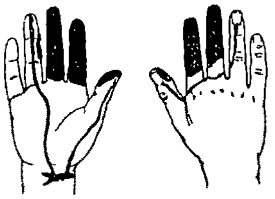


FIG. 31.

To show the extent of the loss of sensation in Case 6.

On March 30, 1904, we noticed the first sign of recovery ; the area insensitive to prick and to the extremes of heat and cold had diminished in size on the index and middle fingers, and had entirely disappeared from the thumb and ring finger. Up to this date, numerous blisters had appeared on the analgesic portions of the index and middle fingers. Some had dried, leaving a mass of thickened epithelium; others had formed shallow ulcers. On March 30, for the first time, the skin was free from any lesion of this character.

By April 27, 1904, all analgesia had disappeared, and water at 60° C. and ice were everywhere appreciated.

The hand remained in this condition, sensitive to prick, but anæsthetic to cotton-wool, until August 10, 1904. The borders of the anæsthetic area were well defined, and within it he failed entirely to discriminate the two points of a pair of compasses separated for a distance of 2 cm.

Shortly after this visit, an abscess appeared at the scar ; it had been opened and had healed when we next saw him on

September 21, 1904. We then found that a change had taken place in the condition of his sensibility. He no longer appreciated a prick and the extremes of heat and cold over the same area, that was insensitive to these stimuli, when we first saw him eleven months before.

On October 23, 1904, he discovered a blister on the terminal phalanx of the middle finger. When we saw him next, four days later, it had broken, and a superficial ulcer marked its site. Sensibility to prick was still lost over the original area, but he occasionally appreciated ice, and water at 50° C.

From this time onward, blisters made their appearance at intervals, usually without any history of injury, and when we saw him on March 30, 1905, the terminal phalanges of index and middle fingers were still analgesic, and ulcers were present. By July 8, these had healed, and the appearance of the skin approached the normal; all analgesia had disappeared.

He still showed a definite line of change to prick on the palm, and within the boundaries of this line he was now sensitive to stimulation with cotton-wool, and the intermediate degrees of temperature. But within this area, he was unable to discriminate two points at a distance of 3 cm.

Case 29.—Injury to the median nerve, produced by a cut at the wrist. Simultaneous return of the two forms of cutaneous sensibility.

On September 20, 1902, whilst loading a van, C. B., aged 27, slipped and cut his left wrist on a broken bottle. He came to the London Hospital at once, and was seen by us, one and a half hours after the accident. He complained of "pins and needles" and "numbness" of the thumb, index, and middle fingers, which had been present since the injury. The wound was oblique, running from the tendon of the flexor carpi radialis, close to the fold of the wrist, upwards and ulnarwards for a distance of 1½ ins. (4 cm.).

The opponens and abductor muscles of the thumb were acting well. He was unable to appreciate light touch over the full median area on the palm and fingers. Sensation to prick and to the extremes of temperature was abolished over the palmar surface of the index and middle fingers and over an area on the palm at their base (*vide* fig. 32). Over the dorsal surface of the two terminal phalanges of the middle finger, the ulnar half of the terminal phalanx of the index and the extreme radial border of the ring fingers, sensibility to prick was also destroyed.

The wound was explored one hour later by one of us, and the median nerve was found to have been injured; it was swollen, and had been cut into on its ulnar side. The tendon of the flexor sublimis digitorum going to the index finger was also divided. After suture of the tendon, the wound was closed and healed by first intention.

On October 22, analgesia was present over the index and middle fingers only, and he was able to appreciate light touch over the proximal part of the palm, both forms of sensation returning together in the manner usual after incomplete division of a nerve. The abductor and opponens pollicis muscles were wasted, but acted readily; they did not react to the interrupted, but reacted normally to the constant current.

By February 11, 1903, he could appreciate a prick everywhere, except over the terminal phalanx of the index and middle fingers; light touch was only lost over these two fingers and the palmar surface of the thumb. All the muscles reacted perfectly to the interrupted current and were no longer wasted.

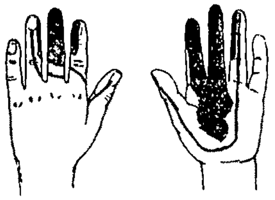


FIG. 32.

To show the extent of the loss of sensation in Case 29.

On July 12, the last occasion on which we saw him, he was able to appreciate a prick everywhere, but the last two phalanges of the index and middle fingers and the palmar surface of the thumb were still insensitive to light touch and the minor degrees of temperature. To the compass test at 2 cm., he showed the phenomenon of "double ones" to perfection.

Case 32.—Division of the median nerve. Improvement after five years.

J. R., a youth of 16, cut his left wrist with glass on February 20, 1897. He was admitted to the London Hospital the same day, and the median nerve was sutured. The wound suppurated and did not finally heal for over four months.

He first came under our observation on January 29, 1902, complaining that "numbness" was still present in the index and middle fingers.

The whole of the affected hand was of a bluish colour, and seemed undoubtedly colder to the touch than the sound hand. The outer thenar group of muscles was slightly wasted, but was acting feebly, and reacted to a strong interrupted current.

The last two phalanges of the index and middle fingers on their dorsal and palmar aspects were insensitive to prick, to ice, and to water at 50° C. Over the usual median area on the palm and the extreme radial border of the ring finger, light touch, the minor degrees of temperature and the interrupted current, applied with no iron in the circuit, were unappreciated. Within this area, he failed to distinguish the points of the compasses, even when separated to a distance of 3 cm.

By August 24, considerable improvement had taken place. There was now no change in the appearance of the skin, all muscular wasting had disappeared, and the contraction evoked by the interrupted current was normal. Analgesia was present only over the terminal phalanx of the middle finger on its palmar aspect. Light touch and the minor degrees of temperature were appreciated over the palm of the hand. Marked improvement had taken place in his ability to distinguish the compass points; he was now perfect at 2 cm., and his answers at 1·5 cm. were good.

By November 16, all analgesia had disappeared, and he could appreciate light touch and the minor degrees of temperature over the whole of the affected parts. This area was bounded by a line of change to prick presenting the usual characteristics; no further improvement had taken place in his ability to appreciate the compass test.

When we last saw him, on January 25, 1903, the condition of the hand remained the same; the blueness and coldness which were present when he first came under observation had disappeared, and had not returned with the advent of winter.

Case 11.—*Division of the median nerve. Trophic changes consisting of blisters, ulcers, and necrosis of the terminal phalanx of the middle finger.*

D. J. T., a stonemason, cut his right wrist with glass in May, 1901. The wound was sutured at once without an anæsthetic.

He first came under our observation on November 27, 1901. An oblique scar ran upwards towards the ulnar side across the lower third of the forearm, from the tendon of the flexor carpi radialis almost to the flexor carpi ulnaris.

The index and middle fingers were the seat of ulcers which had developed as the consequence of infection of what he called "water blisters." On the radial side of the distal phalanx of the index finger, distinct loss of tissue had been produced by a small deep ulcer with indurated edges. On the palmar surface of the same finger were two superficial ulcers, surrounded by a fringe of skin representing the blister from which they had originated. A small blister was present on the dorsum of the second phalanx of the middle finger.

The outer part of the thenar eminence was wasted, and the abductor, and opponens pollicis were not acting; these muscles did not react to the interrupted current and reacted in a typically sluggish manner to galvanic stimulation.

Light touch was lost over the median half of the palm; the palmar surface of the thumb, index and middle fingers, and the radial border of the ring finger, were insensitive to this form of stimulation. The last two phalanges of the index and nearly the whole of the last two phalanges of the middle finger were analgesic.

He remained at his work until May, 1902. During this time the "trophic" changes in his fingers became more pronounced. On April 6, 1902, the last two phalanges of the index and middle fingers were enlarged and the skin thickened. The tip of the index finger was occupied by an ulcer; its nail was represented by a rough, irregular mass. The nail of the middle finger had been cut away to expose an ulcer occupying the tip of the finger, and the nail-bed.

The area of loss of sensation to light touch and to prick remained unchanged on May 14. Two days later, Mr. Eve exposed the nerve and found that it had been completely divided; the ends were lying about 3 cm. apart, united by fibrous tissue. They were freshened and reunited, and the wound healed by first intention (*vide* fig. 3, B, p. 136).

During his stay in the Hospital, the ulcers on the index and middle fingers healed, and the skin regained its normal appearance. He started work again during the last week in July, and a week later a "blood blister" appeared on the tip of the middle finger. By August 6 this had become a shallow ulcer surrounded by thickened epithelium, and on the radial side of the finger was another small dry blood blister. He remained at his work, and the condition of the middle finger became worse; necrosis of the terminal phalanx supervened, necessitating amputation through the second phalanx on November 4.

On December 21 the area of analgesia had become smaller. The general nutrition of the fingers had improved, but there were still small ulcers on the index and middle fingers within the analgesic area.

By January 25, 1903, a prick, ice, and water at 40° C., could be appreciated everywhere within the affected area. All the ulcers had healed, and no further trophic changes made their appearance, although he continued his work as a stonemason.

The opponens pollicis acted voluntarily and responded to a strong interrupted current on May 24, 1903.

The hand remained sensitive to prick and the extremes of temperature, but insensitive to light touch, and water at 22° C. and 38° C., until January 31, 1904. On this date, the borders of the anæsthetic area were as well defined as immediately after suture.

When we again saw him on April 10, he could appreciate light touch and the minor degrees of temperature everywhere over the affected hand. A marked line of change to prick was present, and two points were badly discriminated at 2 cm. He remained in this condition, until we finally lost sight of him in March, 1905.

Case 64.—*Incomplete division of the median nerve. Recovery of the two forms of sensibility* pari passu. *Trophic changes of the nails.*

G. L., aged 13, cut his right wrist with glass in August, 1901. The wound was stitched without an anæsthetic, and the condition of the median nerve was not explored.

He came under our observation on November 4, 1901, complaining of "sore nails."

Ever since the accident he had noticed numbness of the hand, and for a month the nails had been "sore." Three weeks before we saw him, the nails of the index and middle fingers "came off."

A transverse scar was present over the situation of the median nerve, 2·5 cm. above the fold of the wrist. The outer group of thenar muscles was wasted, but acted voluntarily and responded to stimulation with the interrupted current. The terminal phalanges of the index and middle fingers were bulbous, the nails had been shed, and the nail-beds had become ulcers with protuberant granulations forming their floor. The nails of the thumb, ring, and little fingers showed no obvious abnormality.

Cotton-wool, the interrupted current generated without iron in the circuit, and the minor degrees of temperature, were unappreciated over the full median area. Sensibility to prick was lost over the terminal two phalanges of the index and middle fingers. He entirely failed to discriminate two points when separated for a distance of 2 cm.; on a similar portion of the sound palm he made no mistakes at 1 cm.

By February 26, 1902, the area of loss to cotton-wool, the interrupted current, and the minor degrees of temperature, had retreated to the fingers, but the terminal phalanges of the index and middle fingers were still insensitive to prick. He had improved markedly to the compass test, and could discriminate the two points at 2 cm. correctly over the affected palm. The nails had begun to grow, and the nail-beds were no longer ulcerated.

All analgesia had disappeared on April 16, 1902, but he was still insensitive to stimulation with cotton-wool and to the minor degrees of temperature over the last two phalanges of index and middle fingers. A well-marked line of change to prick was present on the palm and on the dorsal surface of the index and middle fingers, bounding the old area anæsthetic to cotton-wool. Within this area he appreciated two points separated to a distance of 1·5 cm. After this date he changed his address and we were unable to see him again.

Case 33.—*Injury to the forearm; Volkmann's contracture. Implication of the median nerve in scar tissue.*

H. E. T., aged 20, fell while playing football on December 26, 1903, and injured his left arm. He was taken at once to an infirmary and his forearm put up in internal and external splints. When these were removed a week later, "a long black bruise" was found over the anterior (flexor) surface of the forearm and a smaller one on the dorsal surface near the wrist. Soon "the bruise began to take a bad turn," the skin broke, and an ulcer appeared, which did not heal for over three months. Fourteen days after the accident "the arm was rebroken and set in a special splint so that the ulcer could be dressed." His forearm remained in splints for three months, and when these were removed, he noticed that the fingers were numb and becoming bent.

When he came under our observation on July 2, 1904, an adherent scar marked the site of the ulcer on the anterior surface of the forearm; over the lower end of the radius, a scar marked the site of the smaller ulcer which had been present there. All the movements of his forearm were free, excepting supination, which was slightly limited. The seat of the fracture was marked by no bony thickening or deformity.

The hand was held a little flexed at the wrist, with the fingers and thumb slightly flexed into the palm. On extension of the wrist, the fingers could not be brought into line with the palm, but when the wrist was flexed they could be almost fully extended. He thus showed the signs of Volkmann's contracture in a slight degree.

The fingers of the affected hand were tapering, and the skin of the palm and fingers smooth, that of the latter being of a reddish-blue colour. A blister, for which he could not account, was present on the palmar surface of the terminal phalanx of the index finger. All the nails of the affected hand "were growing more slowly" than those on the sound side, and showed marked transverse ridges.

The intrinsic muscles of the hand were wasted, but acted voluntarily and reacted to the interrupted current, with the exception of the abductor and opponens pollicis.

He was insensitive to all forms of cutaneous stimulation over the last two phalanges of the index and middle fingers. Light touch and the minor degrees of temperature were not appreciated over the full median area; this was well defined, except at its proximal border. Over the distal portion of the affected part of the palm, he failed to discriminate two points separated for a distance of 3 cm., but over the proximal portion, the formula yielded at this distance was good $(\frac{1 | 6\text{ R. } 4\text{ W.}}{2 | 7\text{ R. } 3\text{ W.}})$. Deep touch and the vibrations of a tuning-fork (C 128) were recognised over the whole of that portion of the hand which was analgesic.

We next saw him on July 24, 1904. During the three weeks that had elapsed, he had received no treatment of any kind, and the nails of the affected hand had grown more slowly than those on the sound side.

	Left (affected).	*Right.*
Thumb	3 mm.	4 mm.
Index	3 mm.	4 mm.
Middle	2·5 mm.	4 mm.
Ring	3 mm.	4 mm.
Little	3 mm.	4 mm.

The appearance of the hand remained the same, but all the muscles now responded to the interrupted current and the outer thenar group reacted to the constant current at 2 ma. with a brisk contraction, K.C.C. appearing before A.C.C.

On August 11, Mr. Dean explored the median nerve in the forearm. The fibrous tissue composing the scar passed deeply, and was adherent to the periosteum of the ulna. In this mass the median nerve was embedded; it was freed and the wound closed. Healing took place by first intention.

The first effect of the operation was to throw back the sensory and motor condition of the hand. The loss of prick became as extensive as when we first saw him, and the abductor and opponens muscles lost their reaction to the interrupted current. No improvement took place for about two months; then both forms of sensation began to return together, and by the end of November, 1904, were only lost over the index and middle fingers. All analgesia had disappeared by February 26, 1905, leaving the terminal phalanges of the index and middle fingers still insensitive to light touch and the minor degrees of temperature. By this date the abductor and opponens muscles had regained their reaction to the interrupted current.

As the result of regular massage, the contracted condition of his fingers had improved considerably and was hardly noticeable on August 27, 1905. At this time he could appreciate light touch and the minor degrees of temperature everywhere, and was perfect to the compass test at 1·5 cm.

Case 19.—*To show the effect of surgical division of the ulnar nerve at the elbow upon deep sensibility and cutaneous sensation of the hand.*

L. C., a tailor, aged 36, came under our care on June 8, 1904. He complained of weakness of the hand, and of pain in the ulnar side, especially troublesome while at work. These symptoms had been present for seven months, and were increasing in severity.

He was unable to appreciate light touch over the full extent of the ulnar area (fig. 33). He could appreciate a prick everywhere, but ice, and water at 50° C. were only recognised over the proximal portion of the palm.

The hand was in the typical ulnar position; all the muscles in the forearm and hand supplied by the ulnar nerve were paralysed and markedly wasted. They did not react to the interrupted current, but responded to galvanic stimulation with a sluggish contraction.

Pronounced changes were present in the lower end of the humerus. The carrying angle of the forearm was much diminished and the internal condyle enlarged and irregular. A swelling was present on the ulnar nerve where it passed between the olecranon and internal condyle, but the nerve could not be displaced. The movement in the elbow-joint was surprisingly good, supination alone being slightly limited. He was not aware of the deformity, and no history could be obtained of any injury during childhood.

From our examination we concluded that the original injury had been a separation of the lower epiphysis of the humerus in early life. It seemed probable that the nerve had been injured as the result of long-continued pressure from the deformed internal condyle. In accordance with this diagnosis, the ulnar nerve was exposed by one of us at the elbow on June 17, 1904. In this situation about 1½ ins. (4 cm.) of the nerve was hard, fibrous, and swollen; 1¾ ins. (4·5 cm.) of the nerve, including the swelling, was removed, after a groove had been made in the bone to prevent the recurrence of the pressure; the ends of the nerve were then sutured. The wound healed by first intention.

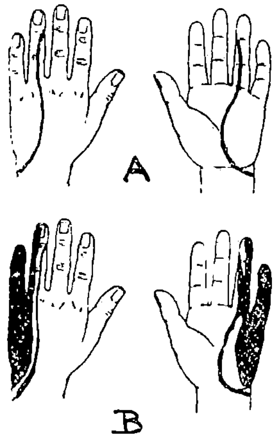


FIG. 33.

To illustrate Case 19.

A shows the area of insensibility to light touch and to the intermediate degrees of heat and cold present before the operation.

B shows the loss of sensation which followed division and reunion of the divided ends of the ulnar at the elbow.

This operation did not increase the area within which he was unable to appreciate light touch, but the extent of the area insensitive to prick was greatly enlarged (fig. 33, B). Deep touch, tested with the head of pin, could not be appreciated over an area a little smaller in all directions than that for loss of prick.

The vibrations of a tuning-fork (C 128) evoked no sensation over the little finger.

By September 14 the analgesic area had diminished considerably in size, and on December 21 occupied the little finger only. Deep touch and the vibrations of a tuning-fork (C 128) were recognised everywhere by May 17, 1905. On August 16, all analgesia had disappeared, and ice could be appreciated over the whole affected area, but he was unable to recognise water at 50° C. over the little finger. The borders of the area insensitive to light touch remained as definite as immediately after section of the nerve.

Case 83.—*Excision of a portion of the ulnar nerve in the forearm.*

In August, 1899, Ernest C., aged 27, cut his wrist severely as he was opening a window. The wound was not stitched, but it healed in a week. About ten days after the accident, pain of a neuralgic character started in the scar, and at last became so troublesome, that in October, 1899, the wound was explored in St. Thomas's Hospital. As the pain did not decrease, he again entered the Hospital on November 30, and on December 21, the ulnar nerve was stretched at the wrist. In February, 1900, the nerve was again explored and freed from cicatricial tissue. As the pain was not materially relieved by these operations, he entered Guy's Hospital in November, 1900. Here the nerve was first stretched, then divided, and, finally, on two separate occasions, a considerable portion was removed in the forearm.

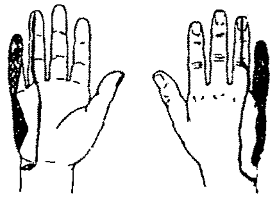


FIG. 34.

To show the loss of sensation produced in Case 83 by excision of a portion of the ulnar nerve in the forearm.

We saw him first on November 20, 1901; at that time the whole hand was wasted, except the muscles over the outer part of the thenar eminence. The fingers were maintained in the position typical of ulnar paralysis. The little finger was blue and cold, and the nails of all the fingers were curved in both directions.

He complained of pain starting over the metacarpal bone of the little finger and travelling along the ulnar side of the hand to the scar on the ulnar aspect of the forearm. This pain he described as neuralgic and shooting in character, not constantly present, and ceasing for forty-eight hours at a time. It was worse in cold weather, and could be started by pressure upon the scar.

Deep touch was lost over the terminal two phalanges of the little finger and over a small area on the ulnar border of the hand. All other forms of sensation were absent over the darkly shaded portion in fig. 34. Cotton-wool was not appreciated over the area enclosed within a single line, and these parts were also insensitive to the interrupted current produced without iron in the circuit and to intermediate degrees of heat and cold. Localisation was extremely defective over the intermediate zone that lay between the border of loss to light touch and that of complete analgesia. Within this zone, the patient was unduly sensitive to all painful stimuli.

The whole of the intrinsic muscles of the hand, excepting the abductor and opponens pollicis, were paralysed, and did not react to the interrupted current. They were so profoundly wasted that we were in doubt whether any reaction was present to the constant current.

Case 34.—*Partial division of the ulnar nerve. Rapid return of both forms of sensibility.*

C. T., aged 28, put his left hand through a glass window on March 4, 1905, cutting the wrist. He immediately felt "pins and needles" in all the fingers of the affected hand.

He came to the London Hospital and the wound was explored four hours after the accident. The ulnar nerve was found to have been cut into below the point where the dorsal branch was given off, and the tendon of the flexor carpi ulnaris had been completely divided. Both nerve and tendon were sutured.

When we saw him on April 8, the wound had healed by first intention, leaving an oblique scar, running from the extreme ulnar border of the wrist upwards and radialwards. The hand was in the position characteristic of ulnar paralysis, and none of its muscles supplied by the ulnar nerve were acting with the exception of the first dorsal interosseous. Stimulation with an

When we next saw her, on July 11, sensibility to prick was everywhere present, and ice, and water at 60° C. were well recognised over the whole affected area. From this time, her power to appreciate temperature deteriorated; on August 8, water at 60° C. was nowhere recognised, and ice was only appreciated on the palm, when the test tube was laid longitudinally and allowed to remain in contact with the skin for some seconds. By November 14, ice was only appreciated over the proximal portion of the palm, although a prick was readily recognised everywhere.

The hand remained in this condition until March 9, 1903. On this date, marked diminution in her power of appreciating prick was noticed, and by May 13 the area of analgesia almost corresponded with the area of loss of light touch. Thus it remained until July, varying, within small limits, from time to time (fig. 35, B).

On July 22, 1903, the condition of the nerve was explored by one of us. It was found to be divided below the point where its dorsal branch is given off, and a remarkable condition was present, effectually preventing all chance of union between its two ends. On tracing the upper portion of the nerve downwards, it was seen to bifurcate; its inner portion had been united to the upper end of the divided tendon of the flexor carpi ulnaris, its outer to the lower end of the same tendon. The lower end of the nerve had been sutured to the divided tendon of the flexor sublimis going to the little finger. The nerve was reunited after its ends had been freshened.

This operation produced no alteration in the distribution or extent of the loss of sensation. On January 14, 1904, all analgesia had disappeared, but she failed to recognise ice over an oval area on the palm, corresponding to that present when we saw her first. Water at 60° C. produced no sensation of warmth over an area almost as extensive as that of the loss of light touch. She could appreciate ice everywhere on March 30, but even on this date water at 60° C. produced no sensation of warmth over the whole affected area, and it was not until June 21 that she was able to recognise this form of stimulation.

Sensibility to light touch began to return over the proximal portion of the palm on March 30, 1904, and by June 27 was lost only over the little finger. It had completely returned by September 21, leaving a well-marked line of change to prick. But on this date water at 40° C. and at 20° C. failed to evoke any sensation of temperature within the line of change, and it was not until May 31, 1905, that these minor degrees were recognised.

On August 16, 1905, the line of change to prick was still present. The compass test was perfect at 2 cm., poor at 1·5 cm. and at 1 cm. out of ten stimulations with two points, a correct answer was given on two occasions only.

On July 27, 1904, the first dorsal interosseous muscle responded to the interrupted current, and acted voluntarily. She is now (August 1905) able to contract all the affected muscles feebly.

Case 63.—*Bullet wound of the ulnar nerve in the forearm. Partial loss of sensation over the ulnar area. Widespread hyperalgesia. Resection and reunion of the two ends of the nerve. Disappearance of all hyperalgesia.*

On July 22, 1901, L. G. H., aged 26, was wounded at Tweefontein; the bullet ricocheted from the butt of his rifle and entered his forearm on the ulnar side in front of the bone, 4½ ins. (11·5 cm.) below the inner condyle of the humerus. It passed across the arm, and was extracted from the radial side eight hours later. When hit, he was retiring with his rifle "at the trail," and it fell from his hand. A severe tingling sensation appeared at once, and he could neither open nor close his hand. After he had been in hospital three weeks, his hand became more painful, and about four weeks after the injury, the excessive sweating and the change in the appearance of the hand was first noticed. During the last two months of 1901, the pain steadily increased; it was worse in a warm room, but if he could keep his hand in either hot or cold water, the throbbing pain ceased. The pain did not keep him awake at night, and never extended higher in the forearm than the wound. The nails began to grow faster than on the sound hand, and that of the little finger grew faster than any other.

Owing to the kindness of Professor Barker, we were permitted to see this man in University College Hospital on January 26, 1902.

He lay in bed with his arm raised, with the radial side of his hand resting on the pillow, so that nothing was in contact with the ulnar half. He was in evident distress, and anxious lest his hand should be touched.

The radial palm and palmar aspect of the thumb, index, and middle fingers were covered with heavy beads of sweat such as is rarely seen upon the hand, even in a Turkish bath, and the skin over these parts had a soft, sodden feeling, as if from fomentations. The ulnar half of the palm and the palmar aspect of the little finger were dry. The whole hand on the palmar surface was smooth and of a pinkish-blue colour; markings were not absent, but they were less numerous and less deep than on the normal hand. On the dorsum the skin was but little affected, but over the dorsal surface of the fingers, particularly over the last two phalanges, the skin was thin and shiny. All the fingers and the thumb tapered from the base upwards, whereas on the sound hand they were more spatulate in form. The nails were curved horizontally and longitudinally, and those of the little, ring, and middle fingers were so painful and tender that he dared not cut them.

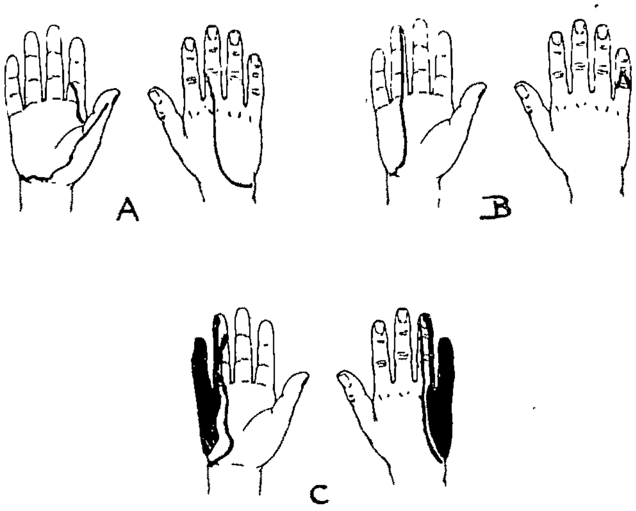


FIG. 36.

To illustrate Case 63.

A shows the extent of the loss of sensation to light touch when the patient first came under our observation.

B shows the area which was intensely hyperalgesic to all cutaneous stimuli causing pain.

C shows the extent of the loss of sensation after excision of a portion of the ulnar nerve and reunion of the divided ends by means of a graft.

He complained of spontaneous throbbing pain mostly in the little, middle, and ring fingers; occasionally the pain invaded the index finger, and it was almost always present over the palmar aspect of the terminal phalanx of the thumb. This pain never extended above the wrist, and did not affect the dorsum of the hand.

He was insensitive to cotton-wool over the ulnar half of the hand, over the little finger, and over the ulnar half of the ring finger (fig. 36, A). The interrupted current, generated without iron in the circuit, was not appreciated over the same area. When 1·5 cm. apart, the two points of the compasses were wrongly appreciated eight times out of ten over the ulnar half of the palm. Over the radial half of the affected hand and over the ulnar half of the normal hand, they were not only perfect at this distance, but were recognised without mistakes when 1 cm. apart.

The whole of the palm of the hand was profoundly hyperalgesic, and if the point of a pin was dragged lightly from the radial side of the thenar eminence towards the ulnar half of the palm he cried out at once that it caused excessive pain when the border marked in fig. 36, B, was passed.

Within the area included by this line, picking up the skin or stimulation with the blunt head of a pin caused intense pain. The borders of this hyperalgesia were difficult to define on the dorsum of the hand, but it seemed to occupy the ulnar half and the whole dorsal surface of the little, middle, and ring fingers. The index finger was not affected, excepting at its extreme base on the ulnar side. Ice, and water at 55° C. were correctly appreciated everywhere. No part of the hand was insensitive to pressure, and within the hyperalgesic area it was uniformly disagreeable.

All the interosseous spaces were profoundly wasted; the muscles of the thenar eminence supplied by the median were unaffected, and he could abduct and oppose the thumb normally. Abduction and adduction of the fingers were impossible; they were out of alignment, and the ulnar two lumbricales were not acting. The thenar muscles supplied by the median nerve reacted well to the interrupted current; but he could stand no current sufficiently strong to test the reaction of the interossei.

On January 30, 1902, Professor Barker exposed the ulnar nerve. The lower end was easily found and was traced up into a mass of firm fibrous tissue where it disappeared; the upper end was lost in the same dense tissue. The two ends were at least 1 cm. apart, and were not in the same direct line. The bullet had apparently injured the nerve in its passage across the limb, and its track was represented by the dense fibrous tissue in which ended both the upper and the lower portions of the nerve; 1 cm. was removed from the upper portion, and the lower end was divided at different levels until healthy nerve fibres were reached. After the track of the nerve had been cleared from fibrous tissue, the two ends were so widely separated, that it was determined to fill the gap with the sciatic nerve of a freshly killed cat. To 5 cm. of this nerve the upper and the lower ends of the ulnar nerve were sutured by means of linen thread, and the forearm was bandaged to a splint with the hand midway between flexion and extension.

He was kept under morphia until the morning after the operation. When he recovered consciousness, he suffered from a good deal of pain in the wound, but all pain and tenderness had left the hand. On February 2, he would permit the hand to be manipulated, and pressure or picking up the skin failed to produce any discomfort.

The whole of the ulnar half of the hand was totally analgesic, the loss of sensation to prick and to the extremes of temperature extending over the ulnar half of the ring finger. The area over which light touch was lost almost exactly coincided with that of loss of sensation to prick (*vide* fig. 36, c).

On March 7, the condition of the hand was that described above, except that the loss of sensation to prick and to the extremes of heat and cold did not extend quite so far towards the radial side; thus there was now a zone of 1·5 cm. in breadth which was sensitive to prick, but insensitive to light touch. By September 3, this had increased considerably. Over this intermediate zone not only was light touch lost, but water at 20° C. and at 40° C. were not appreciated; sensation to prick, to water at 45° C., and to ice was present over this part of the hand. The patient was then compelled to return to his home in Jamaica, and we were unable to examine him further.

Case 84.—Bullet wound of the forearm, causing hyperalgesia over the distribution of the ulnar and internal cutaneous nerves.

In September, 1901, at Blood River Poort, J. D., aged 21, a corporal in Gough's Mounted Infantry, was shot through the left forearm. His arm dropped at once, and the ulnar half of the hand became numb.

The bullet entered 4 cm. below the head of the radius on the outer surface of the forearm, and passed out on the anterior surface, 8 cm. below the internal condyle of the humerus.

We first saw him at Netley on March 2, 1902. He complained of a constant tingling pain, particularly troublesome in cold weather, over the whole ulnar side of the forearm and hand. He had also noticed that the affected portion of his palm sweated more than a similar part of the sound hand.

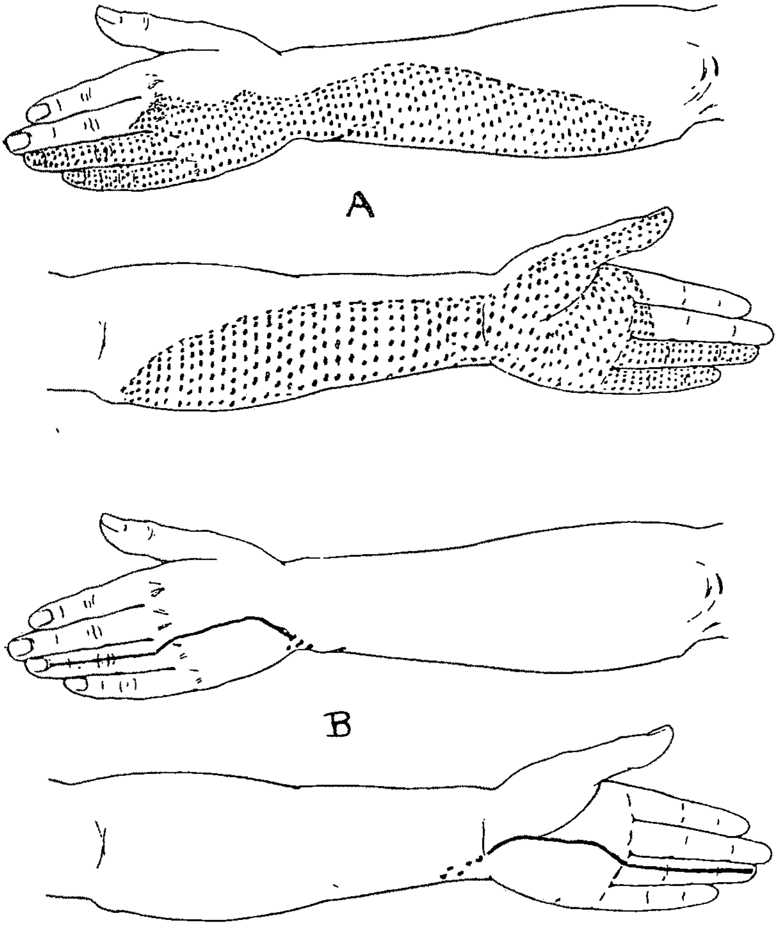


FIG. 37.

To illustrate Case 84.
A shows the extent of the hyperalgesia.
B the extent of the loss of sensation to light touch, and to the intermediate degrees of heat and cold.

Running downwards from the scar on the anterior surface of the forearm was an area of hyperalgesia extending on to the hand (fig. 37). It could be marked out with ease by dragging a point from the sound towards the affected side of the forearm and hand, or by picking up the skin.

On the anterior surface of the forearm, the boundary of the hyperalgesic area ran downwards towards the wrist, a little to the radial side of a line continued upwards from the axis of the ring finger. At the junction of the middle and lower thirds of the forearm, the area extended towards the radial side, reaching the proximal portion of the thenar eminence; from this point it was bounded by a line continued to the radial edge of the thumb-nail. The whole of the palmar surface of the hand and fingers was hyperalgesic with the exception of the index and the terminal two phalanges of the middle finger (fig. 37, A).

On the dorsal (extensor) surface, the boundary of this area of hyperalgesia corresponded in its upper two-thirds to a line continued upwards to the scar from the cleft between the middle and ring fingers. In the lower third of the forearm it swung outwards, reaching nearly to the tendon of the extensor ossis metacarpi pollicis at the wrist. Thence it was continued down towards the knuckle of the middle finger. The whole of the dorsal surface of the hand up to this line, the dorsal surface of little and ring fingers, together with the ulnar two-thirds of the first phalanx of the middle finger, were tender.

Cotton-wool and the minor degrees of temperature could not be appreciated over the full area in the hand supplied by the ulnar nerve (fig. 37, B). Sensation to prick and to the extremes of heat and cold was everywhere present.

There was no marked muscular wasting, and all the muscles acted perfectly and reacted to the interrupted current.

Case 28.—*Division of the median and ulnar nerves, together with several tendons. Primary union of tendons. Secondary suture of the nerves after the tendons had healed. Condition of deep sensibility.*

G. B., a carpenter, aged 24, cut his right forearm with glass on September 24, 1902. The wound was explored the same day without an anæsthetic; several divided tendons were sutured, but the nerve injury was not discovered. Two or three days later he noticed that he had "lost all feeling" in the palm of the hand and fingers.

He was admitted to the London Hospital on April 15, 1903, and we saw him the following day. He had been at work for five weeks; a week after he had begun work a blister had appeared on the fingers, which had burst and discharged, leaving an ulcer.

In order to treat this condition he placed the fingers in hot water, and so produced further blisters. This resulted in the following condition, which was present when we first saw him. A superficial ulcer occupied the surface of the last two phalanges of the index finger; the nail had disappeared, leaving a granulating surface discharging pus. The terminal phalanx of the middle finger was in a similar condition. No blisters had appeared on the other fingers, although the whole hand had been placed in the hot water.

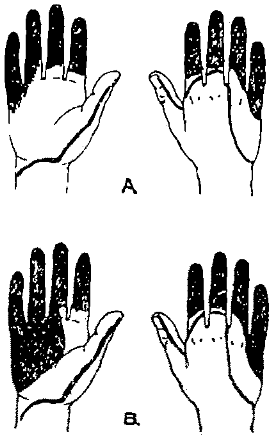


FIG. 38.

To illustrate Case 28.
A shows the loss of sensation before the operation.
B shows the loss of sensation produced by the operation of April 17.

A flap-shaped scar was present on the anterior surface of the forearm, extending downwards and radialwards from 8 cm. above the head of the ulnar to 3 cm. above the fold of the wrist at the tendon of the flexor carpi radialis; here it changed its direction and ran almost vertically upwards for 3 cm.

The hand was in the position typical of ulnar paralysis, and all its intrinsic muscles were wasted and paralysed; they did not respond to stimulation with the interrupted current.

Sensibility to light touch and to prick were lost over the areas in fig. 38, A.

On April 17 the condition of the nerves was explored. The median was found to have been completely divided and its ends separated, the lower being united to a tendon. The upper end of the ulnar was bulbous and united to the lower by a thin strand of tissue. Both nerves were reunited after the ends had been freed and freshened.

This operation resulted in a considerable increase in the area insensitive to prick and to the more extreme degrees of heat and cold (fig. 38, B).

No improvement in the sensory condition of the hand took place until after August 23. The area insensitive to light touch and to prick remained as extensive and well defined as immediately after suture. He failed to appreciate ice, and water at 50° C. over the area insensitive to prick, and over the area anæsthetic to cotton-wool he could not recognise water at 22° C. and at 40° C. Sensibility to deep touch was present and well localised everywhere except over the little finger. To the compass test applied over the median half of the palm, he failed entirely at 3 cm., but when the two points were applied successively, he made no mistakes at 2 cm. and was rarely wrong at 1·5 cm.

By September 27, he could appreciate a prick and the more extreme degrees of temperature over the whole of the palm and dorsum of the hand, the fingers still remaining insensitive to these stimuli. By December 20, all analgesia had disappeared and he could appreciate stimulation with cotton-wool over the dorsum of the hand, but if this area was shaved, it was found to be entirely insensitive to this stimulus.

Case 43.—Complete division of the musculo-spiral nerve.

F. L., a boy of 9, fractured the lower end of his right humerus on June 28, 1903. Two days later an open operation was performed in order to reduce the deformity.

Paralysis of the muscles of the forearm supplied by the musculo-spiral nerve resulted from this operation. In consequence of this condition, he came under the care of one of us at the Poplar Hospital for Accidents on July 28, 1903.

All the muscles in the forearm supplied by the musculo-spiral nerve were wasted and paralysed; they did not react to the interrupted current, but responded with a characteristically

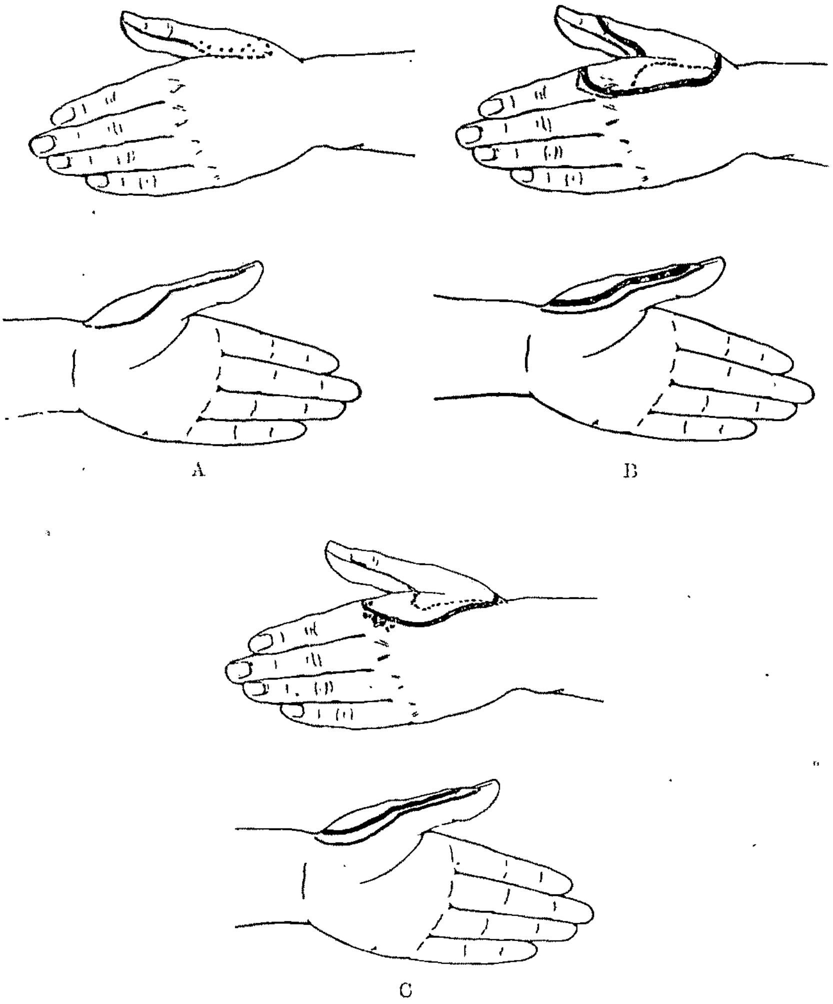


Fig. 39.

To illustrate Case 44.

A shows the area which became insensitive to light touch in consequence of division of the radial nerve (r. superficialis n. radialis) at the wrist.

B shows the areas of loss of sensation produced by subsequent division of the posterior branch of the external cutaneous nerve at the elbow. The thick line encloses the parts insensitive to prick, the thin line the parts insensitive to cotton-wool.

C shows the additional loss of sensation on the thumb caused by subsequent division of the branch of the median nerve to the ulnar half of the thumb.

ciated everywhere. The tender area on the dorsum of the thumb was still present, although the tenderness was less marked. Consequently, on September 9, 1904, the posterior division of the external cutaneous nerve was divided at the bend of the elbow. This operation increased slightly the area insensitive to light touch on the dorsum of the hand, but left it unaltered on the thenar eminence and thumb. A prick and the more extreme degrees of temperature could not be appreciated over an area almost as extensive as that within which light touch was lost, but a small area of dissociated sensation was present on the dorsum of the hand sensitive to cotton-wool, but insensitive to prick and to all degrees of temperature (*vide* fig. 39, B). Deep touch was appreciated over the whole of the affected area. No loss of sensation resulted in the forearm from this operation.

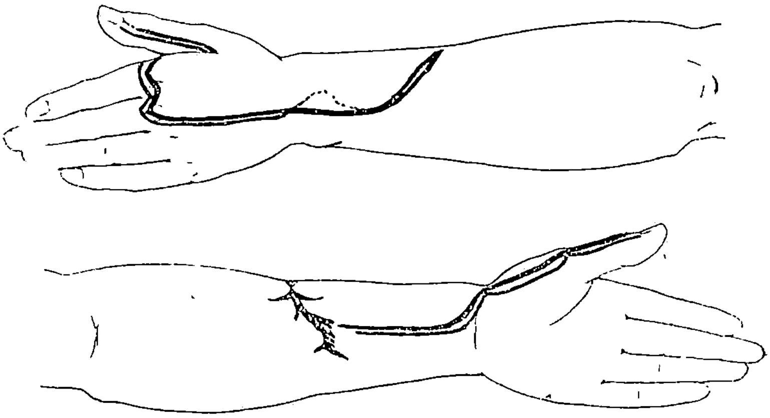


FIG. 40.

From Case 47, to show the area of loss of sensation produced by the accidental division of the radial (r. superficialis n. radialis) and external cutaneous nerves. The jagged scar is represented running across the flexor surface of the forearm. The area insensitive to light touch is enclosed by a thin line, that insensitive to all cutaneous stimulation by a heavy black line.

As a little tenderness still remained at the extreme ulnar border of the tip of the thumb, the branch of the median nerve supplying the inner side of the thumb was divided on October 5, 1904. This rendered the ulnar border of its terminal phalanx entirely insensitive to all forms of cutaneous stimulation (fig. 39, C).

Case 47.—Accidental division of the radial (r. superficialis nervi radialis) and external cutaneous nerves in the forearm. The case illustrates the characteristics of deep sensibility.

G. S., a cabinet-maker, aged 23, cut his left forearm with a broken window on July 23, 1903. The wound was sutured immediately, but suppurated severely, and he was admitted to the London Hospital with secondary hæmorrhage ten days later.

He first came under our observation on October 25, 1903.

On the radial border of the lower third of the forearm was a multiradiate scar, its centre situated 9 cm. above the wrist, depressed and adherent to the underlying structures.

All the movements of the hand and fingers were perfect, and there were no obvious changes in the appearance of the skin.

He was unable to appreciate a prick, light touch, and all degrees of temperature over an area on the forearm and hand shown in fig. 40. The boundary of the loss of each of these forms of sensibility was identical, except over a triangular area situated just above the wrist. Over this portion of the dorsum of the hand, measuring 5 cm. in length and 4 cm. in breadth at its widest part, cotton-wool was definitely appreciated, but sensibility to all degrees of temperature was abolished.

Deep sensibility was present over the whole of the affected area, and he localised well the point of application of the stimulus. He readily appreciated stimulation with cotton-wool rolled up into a pledget and applied vertically, or dabbed on to the hand. But he entirely failed to distinguish between the point of a pin and pressure with a steel rod 2 cm. in diameter. If the skin was raised from the underlying structures, he lost all power of appreciating pressure.

Over the affected area on the dorsum of the hand, he was unable to discriminate two points at 5 cm., applied simultaneously in a longitudinal direction. But when the points were applied successively, he frequently recognised the double nature of the stimulus, although the points were only 2 cm. apart. Over a similar portion of the sound hand he made no mistakes at 3 cm., and only two at 2 cm.

We had the opportunity of examining this patient on many subsequent occasions. The area of loss of all forms of cutaneous sensibility remained unaltered, and the triangular area of dissociated sensibility persisted; but the amount of sensation evoked by cotton-wool varied considerably, being less marked in the winter.

When we last saw him on September 11, 1905, this area was as well marked as when we first tested him, and the character of its sensibility remained unchanged. After shaving, it became entirely insensitive to cotton-wool, and the area of loss to this form of stimulation then corresponded exactly to that of the loss of prick.

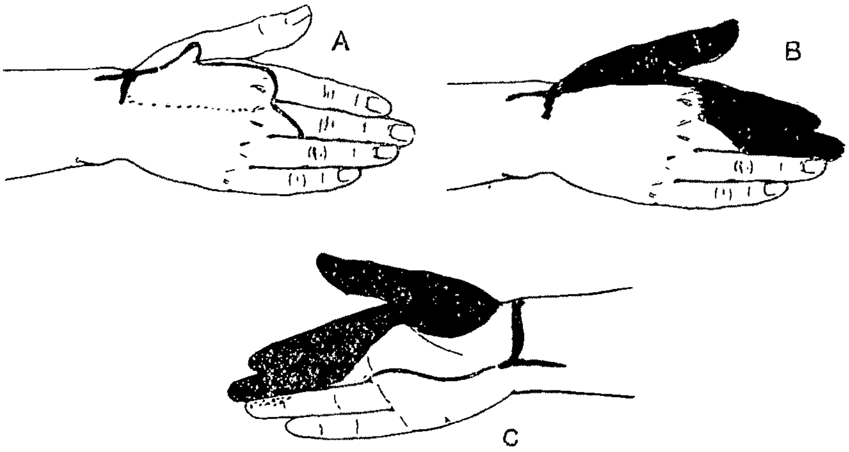


FIG. 41.

To illustrate Case 85.

A represents the area insensitive to light touch; the dotted line enclosed an area of diminished sensibility.

B shows the parts insensitive to prick and to heat and cold.

C shows the parts insensitive to prick in black and those insensitive to light touch enclosed by a line.

Case 85.—Division of the median, radial and part of the external cutaneous nerves. Trophic changes in the skin of the insensitive fingers. Dissociated sensibility on the radial side of the back of the hand.

T. S., a plumber, aged 30, was admitted to the London Hospital on May 12, 1902.

Six weeks previously (March 29) he had put his right hand through a glass panel, cutting the wrist. The wound was sewn up the next day, and "never properly healed." He noticed that the radial half of the hand was numb immediately after the accident.

A transverse scar ran round the radial portion of the wrist from the base of the third metacarpal bone on the back of the hand to the second on its palmar surface. In the centre of this wound was a small granulating area.

The hand was held adducted to the ulnar side and slightly flexed. The outer group of thenar muscles was wasted, and the abductor and opponens pollicis were not acting and did not respond to stimulation with the interrupted current. The flexor and extensor tendons of the thumb, the flexors of the index finger, and the radial extensors of the wrist, were seen to be divided on throwing the muscles into action.

The skin over the whole palm was desquamating; the middle and index fingers were covered with rough scales, but the little and ring fingers had already completely desquamated.

The boundaries of the area of loss of sensation corresponded almost exactly on the palm for all forms of cutaneous stimulation, and the borders were well defined (*vide* fig. 41). On the dorsum, over the distal portion of the first interosseous space, light touch, though diminished, was appreciated over a small triangular area where he was entirely insensitive to prick and all degrees of temperature.

The boundary of the loss to light touch was here ill-defined, and the area of loss merged on the ulnar side into parts of normal sensibility.

On June 3, the median and radial nerves were exposed. They were found to have been divided, and were reunited.

No change in the sensory condition of the hand resulted from this operation. It was not until November 16, 1902 (166 days after suture), that the analgesia had retreated from the palm. By September 6, 1903, he could appreciate a prick and the more extreme degrees of temperature everywhere over the affected hand except the terminal phalanges of the index and middle fingers. The area insensitive to cotton-wool remained as extensive and well defined as before suture.

Up to this date, burns and injuries arising during his work as a plumber had resulted in the formation of ulcers, but with the return of sensibility to prick all the ulcers had healed and none appeared subsequently.

By December 20, 1903 (565 days), the area on the palm and dorsum of the hand anæsthetic to light touch had diminished in extent, and on January 31, 1904, occupied only the last two phalanges of the fingers and terminal phalanx of the thumb; water at 24° C. and at 40° C. were appreciated except over these parts. To the compass test at 2 cm. he made only two mistakes, but 1·5 cm. was obviously below the threshold.

On February 24, 1904 (634 days), the opponens and abductor reacted to the interrupted current, but no voluntary movement was observed until August 28. At this time he was still insensitive to cotton-wool and the intermediate degrees of temperature over the terminal phalanges of the index and middle fingers.

Case 86.—Bullet wound of the ulnar and musculo-spiral nerves. Hyperalgesia over the peripheral distribution of both nerves. Loss of sensation to light touch and to the intermediate degrees of heat and cold over the full ulnar area.

R. M., a corporal in the Imperial Yeomanry, serving in South Africa, was shot through the left shoulder at fifty yards, on November 25, 1901.

He immediately felt as if he had received "a sharp blow on the shoulder" and found that his arm was numb and useless. He fainted and, falling from his horse, lay on the veldt for about two hours. When he regained consciousness, he had so far lost all sense of the position of the limb that, as he told us, he "felt for it here and it was there."

Seven days after the injury, the shoulder began to swell and "was swollen to several times its usual size for four or five days." Ten days after he had been wounded, pain began in the shoulder, and "gradually worked down the arm, leaving the shoulder free, settling in the hand." The wound was completely healed in three weeks.

He came under our observation at the Royal Victoria Hospital, Netley, on March 24, 1902, and again in August of the same year. No change took place in his condition during this time.

The wound of entry was situated at the junction of the arm with the anterior axillary fold 7·5 cm. below the coracoid process. The wound of exit lay over the inferior border of the scapula, 11·5 cm. below its spine. All the muscles of the shoulder and arm were wasted, and the biceps being less affected than the others stood out prominently. All except the triceps were acting voluntarily. The muscles of the forearm were wasted and the wrist was dropped; all power of extension of the wrist and fingers was lost and the supinator longus was not acting. All the flexor muscles acted feebly. The whole hand appeared wasted, and the muscles supplied by the ulnar nerve were paralysed.

The biceps, extensors of the wrist and fingers, supinator longus, interossei and adductors of the thumb did not react to the interrupted current.

The skin of the affected palm was smooth, pink and mottled. The little finger and ulnar border of the hand were blue and wrinkled. All the fingers tapered and the nails were kept long on account of the pain caused by cutting them. They were smooth, and showed excessive curving, both transversely and longitudinally.

He complained of pain in the hand "as if it were going to burst," and said: "I have often looked at it, fancying it must be bleeding." The pain was continuous, and he could only obtain relief by rubbing it with olive oil, which eased him for about an hour.

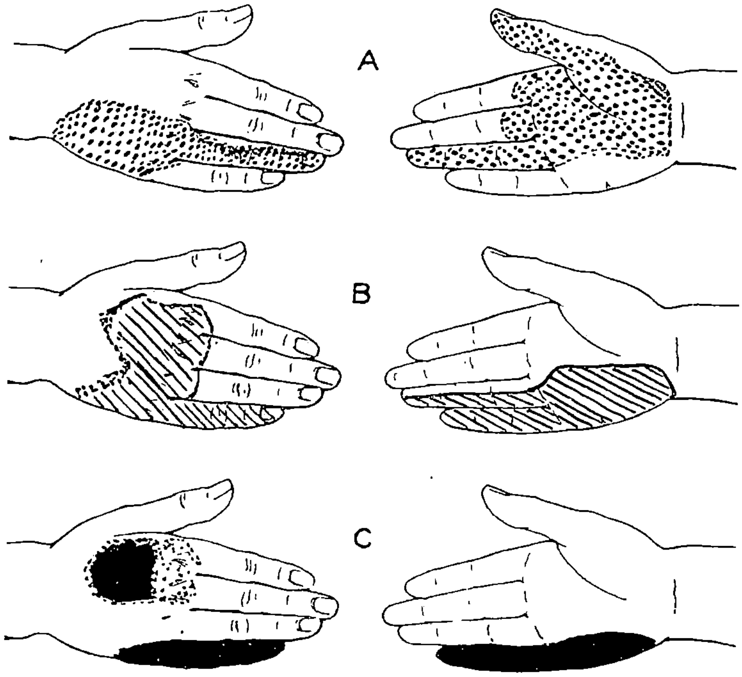


FIG. 42.

To illustrate Case 86.
A shows the extent of the hyperalgesia.
B shows the area insensitive to light touch.
C shows the area insensitive to prick and to all degrees of heat and cold.

Extreme hyperalgesia existed over the area shown in fig. 42, A. Sensibility to cotton-wool was absent over the full ulnar area, which merged on the dorsum, by means of a band of diminished sensibility into an area on the radial side within which he usually failed to recognise a touch with cotton-wool. Water at 22° C. and at 38° C. were not appreciated over that portion of the hand where light touch was affected. He was unable to appreciate prick, ice, and water at 55° C. over the area shown in figure 42, B.

After leaving Netley on August 26, 1902, he attended a civil hospital until February, 1904, and was treated with massage and galvanism. All pain and tenderness gradually disappeared.

When we saw him again on April 3, 1905, all the muscles of the upper limb were wasted. Those on the extensor surface of the limb acted voluntarily except the extensor longus pollicis, and all responded to stimulation with the interrupted current.

None of the intrinsic muscles of the hand acted, or reacted to the interrupted current.

All trace of hyperalgesia had disappeared, and he was able to appreciate a prick and the extreme degrees of temperature over the whole of the affected hand. He was anæsthetic to cotton-wool over the ulnar area on the palm, but appreciated it everywhere on the dorsum, even after the hand was shaved.

Case 64.—*Bullet wound of the arm injuring the musculo-spiral, median and internal cutaneous nerves. True hyperalgesia. Two forms of cutaneous trophic change.*

James W., aged 23, a corporal in the Imperial Yeomanry (Royal Victoria Hospital, Netley). On August 1, 1901, he was shot through the left arm with a Martini bullet. He fainted and fell from his horse, but suffered no pain. The Boers came up, took what they wanted from him, and left him lying for two days and one night on the veldt. He was then taken to hospital, but the "wound became very foul," and did not heal for more than five weeks.

At first the arm was entirely painless; but about a month after the injury the hand began to be painful. The pain steadily increased until it became constant. It did not vary to any considerable extent except in cold weather. In the winter, it was scarcely troublesome so long as the hand was exposed to cold, and cold water always removed the pain for a time.

We first saw him on March 3, 1902. He was then a well-built, healthy looking man, with a somewhat anxious expression. He carried his arm in a sling with the hand exposed, and was evidently terrified lest it should be touched or jarred.

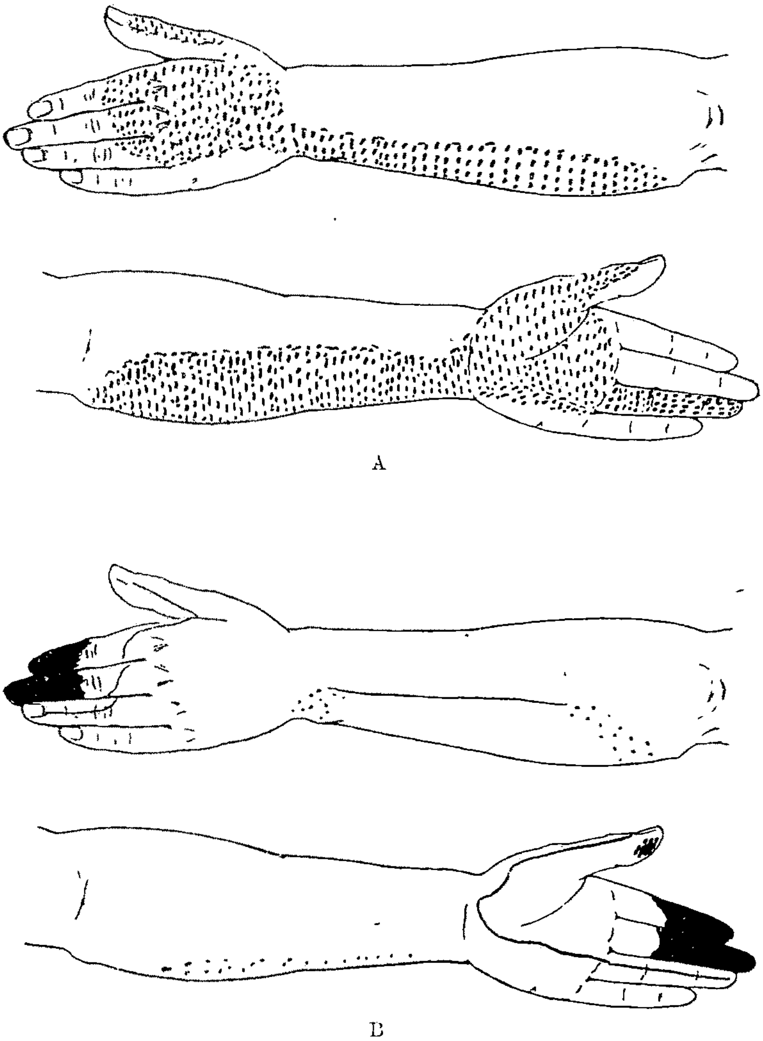


FIG. 43.

To illustrate Case 64.
A shows the area that was hyperalgesic.
B shows the area insensitive to light touch enclosed in a single line. The parts insensitive to prick and to all degrees of heat and cold are coloured black.

The wound of entry was situated 1½ ins. (4 cm.) above the external condyle of the humerus. It was triradiate in shape, measuring 1¼ in. (3 cm.) by ½ in. (1·2 cm.). Two and three-quarter inches (7 cm.) above the internal condyle was the wound of exit, oval in shape, 1 in. (2·5 cm.) by ¾ in. (2 cm.) in size. Both wounds showed a tendency to keloid formation.

The whole of the left hand was smooth and of a bluish-pink colour, the two terminal phalanges of the index and middle fingers were cold and blue. The radial half of the palm and the index and middle fingers did not sweat, whereas the whole of the remainder of the palm was sweating profusely.

The muscles on the extensor surface of the forearm and the outer group of the thenar muscles were much wasted. No extension was possible at the wrist, but, with the fingers extended, the hand could be raised to the horizontal. The supinator longus was acting well. All movement was absent in the abductor and opponens pollicis. All the muscles supplied by the ulnar nerve reacted perfectly to the interrupted current, but no reaction was obtained from the opponens and abductor pollicis. Neither of the extensors of the wrist nor those of the fingers reacted to the interrupted current, but all the remaining muscles of the forearm responded. The extensors of the fingers acted feebly, but failed to respond to faradic stimulation. The muscles supplied by the median nerve neither acted voluntarily nor reacted to the interrupted current.

He complained of great tenderness over the ulnar half of the palm of the hand, and over the greater part of the dorsum. The head of a pin caused considerable pain when the limits of the area in fig. 43, A, were reached. Sensibility to cotton-wool was lost over an area that occupied the ulnar half of the forearm on the dorsal surface. To the radial side, this anæsthetic strip was sharply marked off from the remainder of the arm, but the proximal and distal ends, and the limits of loss to light touch on the front of the forearm, were indefinite, merging gradually into parts of normal sensibility. On the palm of the hand and over the fingers, light touch was lost within the area shown in fig. 43, B, corresponding to that commonly seen after division of the median nerve. Sensation to prick was absent from the index and middle fingers over two and a half phalanges on the palmar aspect, and the two terminal phalanges on the dorsal surface. Water at 40° C. was not appreciated over the area where light touch was lost on the forearm and palm of the hand. Ice, and water at 50° C. produced a sensation of cold and of heat, excepting over those parts of the fingers insensitive to prick. Tested with the compass points over the normal palm and over the ulnar portion of the affected palm, the record was perfect at 1·5 cm. Over the radial half of the palm of the affected hand within the limits insensitive to light touch, but sensitive to prick, ten stimuli with the two points at 3 cm. were called "one"; right answers were given in every case when one point only was used. By this test there was no material diminution of sensation over the ulnar half of the affected palm, but over the median half the points at 3 cm. were evidently below the threshold of sensation.

On March 27, 1902, we saw him again and found that a sore had made its appearance over the terminal phalanx of the middle finger; it first appeared as a blister without known cause, and was entirely painless. It had all the appearances of a trophic sore of a kind not infrequently seen over totally analgesic parts. We were told by the medical officer in charge of the case that in December, 1901, a herpetiform rash had made its appearance over the ulnar half of the palm of the hand, and part of the little and ring fingers, that is to say, within the area of true hyperalgesia. Thus, this man showed both forms of trophic-lesion of the skin which make their appearance in consequence of injuries to nerves.

On our first visit we had noticed that the nails of the thumb, index and middle fingers were abnormally curved, both in a transverse and longitudinal direction. He stated that these nails did not grow so quickly as those of the normal hand, and this statement was borne out by measurements made at our second visit. In the twenty-four days which had elapsed, the growth of the nails of the two sides was as follows:—

	Sound Hand.	*Affected Hand.*
Thumb	5 mm.	3 mm.
Index	6 mm.	1·5 mm.
Middle	6·5 mm.	4 mm.
Ring	5 mm.	5 mm.
Little	4 mm.	4 mm.

Thus, although the nails of the ring and little fingers showed an equal amount of growth on the affected hand, those of the index and middle fell far behind.

We were able to confirm our previous observations on the limits of the hyperalgesia, and loss to light touch. The extent of the loss to prick was somewhat less than on our first visit.

Case 57.—Bullet wound of the great and small sciatic nerves.

J. W. B., a private in the 1st Manchester Regiment, was wounded at Witklip on October 4, 1901. He was ambushed when on water picket and shot from a distance of about twenty yards. He at once felt great pain down the back of the leg. The wound was dressed half an hour later, and he was taken to Leidenberg Hospital the same day.

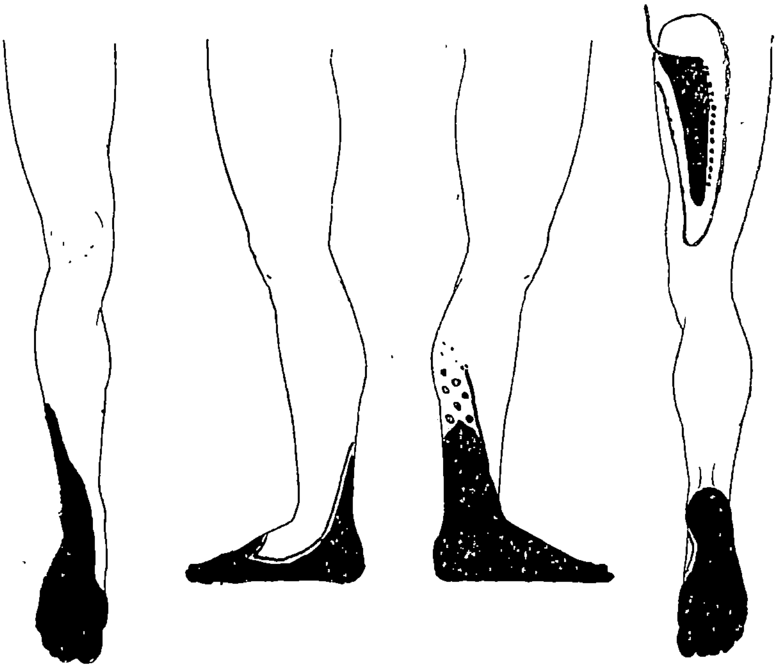


FIG. 44.

To illustrate the loss of sensation produced in Case 57 by an injury to the great and small sciatic nerves. Total loss of cutaneous sensibility is represented in black. Loss of sensation to light touch is enclosed by a line.

He came under our observation at Netley in August, 1902. The wound of entrance, situated 2·5 cm. to the right of the spine of the third sacral vertebra, had not yet healed; 4 cm. anterior to the upper border of the great trochanter was a small surgical scar through which the bullet had been extracted,

All the muscles of the lower limb supplied by the sciatic nerve were wasted, and, with the exception of the hamstring muscles, were paralysed. None of the muscles supplied by the sciatic nerve contracted to the interrupted current, and to the constant current all reacted sluggishly. These muscles responded more easily to the positive than to the negative pole, but no reaction could be obtained from the hamstring muscles, even with a current sufficiently strong to cause contraction in the muscles on the anterior surface of the limb.

Extending downwards from the fold of the buttock for about 40 cm. was a strip, within which he was unable to appreciate prick and the more extreme degrees of temperature. Surrounding this in every direction, and extending on to the buttock, was an area anæsthetic to cotton-wool (fig. 44).

Over the greater part of the foot and outer portion of the lower third of the leg, light touch, prick, and all degrees of temperature were unappreciated; the borders of loss of sensation to each of these stimuli were almost exactly co-terminous.

This area was continued upwards into parts insensitive to cotton-wool, and to intermediate temperatures, but sensitive to prick, and to the more extreme degrees of heat and cold. Above the loss of sensation merged gradually into parts of normal sensibility.

REFERENCES.

(1) BEEVOR. "The Croonian Lectures on Muscular Movements and their Representation in the Central Nervous System." Delivered June, 1903 (London, 1904).

(2) BOWLBY. "Injuries and Diseases of Nerves." London, 1889.

(3) BROOKS. "On the Distribution of the Cutaneous Nerves on the Dorsum of the Human Hand." *Internat. Monatschrift f. Anat. u. Physiolog.*, 1888. Vol. 5.

(4) DENMARK. *Medico-Chirurgical Trans.*, 1813. Vol. 4.

(5) HAMILTON. *Dublin Medical Journal*, 1838.

(6) HÉDON. "Etude Critique sur l'innervation de la face dorsale de la main." *Internat. Monatschrift f. Anat. u. Physiolog.*, 1889. Vol. 6.

(7) KENNEDY. "On the Regeneration of Nerves." *Phil. Trans. Roy. Soc.*, 1897. Vol. 188, Series B., p. 257.

(8) LÉTIÉVANT. "Traité des Sections Nerveuses." Paris, 1873.

(9) MCDOUGALL. *Reports Cambridge Anthropological Expedition to Torres Straits.* Cambridge, 1903. Vol. 2, Pt. 2, p. 189.

(10) PAGET. "Some Cases of Local Paralysis." *Medical Times and Gazette*, 1864. Vol. 1, p. 331.

(11) SHERRINGTON. "Examination of the Peripheral Distribution of the Fibres of the Posterior Roots of some Spinal Nerves." Part I. *Phil. Trans. Roy. Soc.*, 1892. Vol. 184, Series B., p. 641.

(12) SHERRINGTON. "Examination of the Peripheral Distribution of the Fibres of the Posterior Roots of some Spinal Nerves." Part II. *Phil. Trans. Roy. Soc.*, 1898. Vol. 190, Series B., p. 45.

(13) SHERRINGTON. "On the Spinal Animal." *Medico-Chirurgical Trans.*, 1899. Vol. 82.

(14) WEIR-MITCHELL. "Injuries of Nerves." Philadelphia, 1872.

["Gunshot Wounds and other Injuries of Nerves," by Weir-Mitchell, Morehouse and Keen, Philadelphia, 1864, we have not seen. This work is frequently quoted in (14).]

(15) ZANDER. "Ueber die sensibeln Nerven auf der Rückenfläche der Hand bei Säugethieren und beim Menschen." *Anat. Anzeig.*, 1889. Vol. 4.

THE STRUCTURE AND FUNCTION OF THE TASTE-BUDS OF THE LARYNX.

BY JOHN GORDON WILSON, M.A., M.B.EDIN.,
From the Hull Anatomical Laboratory of the University of Chicago,

With 2 Plates : Plates II. and III.

On the minute anatomy of the taste-buds of the tongue much work has been done, but relatively little on the buds of the larynx. Yet in this cavity, on account of the isolated position of the buds, their structure can be more easily discerned and the nerve supply more closely studied than in the mouth. The investigations which are described in this paper have been chiefly directed toward the morphological characteristics of the laryngeal buds, especially the nerve supply, including the nerve-endings. It was hoped that such investigations would throw some light upon the physiological action of taste-buds in general and upon any additional physiological action which the taste-buds of the larynx might possess.

The tissue examined was obtained from man, from the monkey, dog, and rabbit. The method employed was chiefly the *intra vitam* injection of methylene blue, as described in a former paper (Wilson, 1904). The tissue was usually counterstained with orange G acid fuchsin. At the same time sections were stained by the Golgi method, by iron hæmotoxylin, and by gentian violet.

In the larynx the taste-buds, first described by Verson, in 1871, are found on the laryngeal surface of the epiglottis and the medial and lateral surfaces of the arytenoids. They are usually met with in groups, but never closely packed together as in the tongue. Buds are found only in the squamous epithelium. They do not project beyond the surface, but lie in a very shallow saucer-like depression (Shofield, 1876).

They extend through the whole depth of the epithelium, and each rests on a broad papilla of relatively dense connective tissue, through which vessels and nerves enter the buds. These papillæ are not always prominent, and may be absent; when present, they are easily observed, since over the cartilage of the epiglottis the epithelium possesses relatively few papillæ.

Notwithstanding the shallow saucer-like depression of the epithelium in which the buds lie, their pores are very much exposed, and so are in marked contrast to the concealed nature of the taste-pores of the tongue (Plate II. A). Each bud is surrounded, except at its base, by a wall made up of epithelial cells, which are flattened laterally and imbricated (fig. 1; compare extrabulbar cells of Gräberg, 1899). A

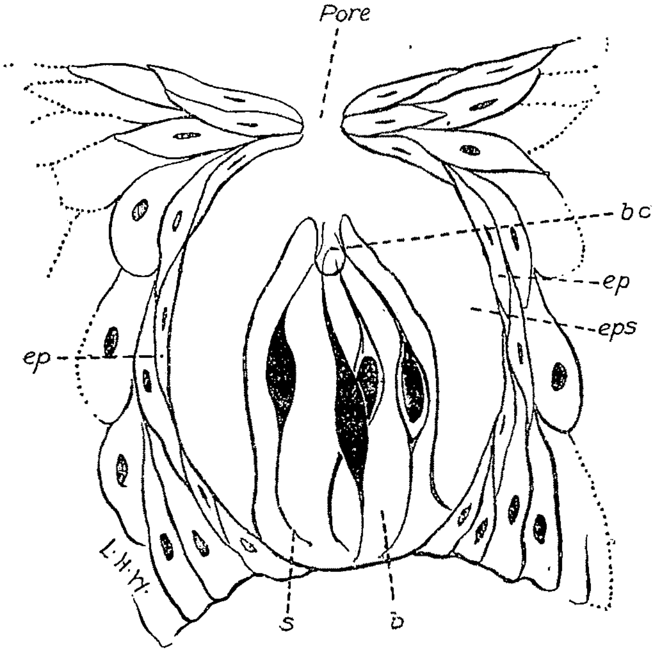


Fig. 1.

Section of the epiglottis of a dog, showing the arrangement around the bud of the epithelial cells—flattened and imbricated (*ep*). Taste-bud (*b*) retracted from the sides, but attached at base leaving a space (*eps*). Within the bud four spindle-shaped cells (*s*) project into bud cavity (*bc*).

Zeiss $\frac{1}{12}$ *oil immersion. Comp. occ. 4.*

condition of cornification can be observed in many of them. They fit closely to each other, and are adapted to the shape of the bud. Towards the surface the cells are so adjusted as to form a clean-cut edge around the pore, sloping slightly in a radial direction to it. The pore proper is formed entirely by these epithelial cells. Towards the subepithelial tissue the cells have their bases flattened and so extended as to embrace the papilla. There is here, therefore, such a modification of the epithelium adjacent to the bud that it may be regarded as forming a protective capsule, the closely packed epithelial cells being in marked contrast to the loosely arranged cells of the bud. To this capsule the bud is attached by protoplasmic bands, and out of this epithelial setting, after fixation, it easily drops.

The laryngeal taste-bud, like that of the tongue, has two types of cells, the spindle-shaped cell and the supporting cell, also a flasked-shaped cavity in its upper segment.

(1) The spindle-shaped cell (fig. 1 and Plate II. B) extends the whole length of the bud; it is not confined to the axial region. Its central termination, long, fine, and unbranched, reaches to the connective tissue of the papilla, but not into it. Peripherally its cytoplasm is prolonged into a very fine hairlike process which projects into the cavity of the taste-bud, and may even reach to the taste-pore. In vital staining with methylene blue, not only does the nucleus stain readily, but the facility with which the whole cell takes up the dye is in marked contrast to the non-reaction of the supporting cell and of the surrounding epithelial cells to the dye. In these cases the cell is very sharply outlined.

The evidence in support of the special sense function of the fusiform cell is based on :—

(*a*) The fact that it colours readily with neurotropic dyes, as opposed to the non-colourability of the supporting cell.

(*b*) The morphological character of the cell.

(*a*) Primary colourability as a test for a neuro-epithelial cell, though of great value, is subject to so many exceptions that alone it is open to objections. Methylene blue, although neurotropic, is not monotropic (Ehrlich, 1902). It is impossible to deduce from primary colourability that we are dealing with a nerve termination. Some cells which so stain cannot be recognised as neural cells; for instance, one cell in the epithelium which readily takes up the dye is the so-called "wandering cell," a cell which has been mistaken for and described as a ganglion cell (Dogiel, 1903). In the bud, however, we have two varieties of cells, one of which, the spindle-shaped cell, shows the neurotropic character, the other does not; so we are justified in assuming that the former possesses a certain chemical constitution which allies it more closely to the nerve-cell.

(*b*) When to this we can add such morphological characteristics as its peripheral process with its relation to the pore, its analogy to other special sense-cells, we have support for the belief that the fusiform cell is specially engaged in the mediation of the sense of taste.

(2) The second type of cell, the so-called supporting cell, differs in no respect from the corresponding cells in the buds of the tongue. Its shape varies very greatly, but there are not two types of this cell, one peripheral and one central. The irregularity is due in some degree to fixation. The cells are loosely applied and adjusted to each other, to the taste-cells, and to the epithelium around, leaving spaces through which leucocytes may wander (Ranvier, 1888). Under fixation they readily shrink and may withdraw from each other, but chiefly as a bud from the epithelial wall from which they may separate at any point. When this shrinkage occurs, the intercellular bridges which pass from the epithelial wall to the supporting cells are easily observed and without doubt distort the cell wall. While this is so, the general relation of the supporting cell to the taste-cell and its general outline in cross-section prove that we have to do with a cell which readily adjusts its contour to the surrounding parts. In the centre of the bud it appears somewhat triangular, with the fusiform sensory cell applied to the flattened and slightly concave surface; at the periphery there is a similar adjustment of the external surface to the epithelial wall—convexity to concavity. Its general appearance and its mobility of surface suggest a cell protoplasm easily susceptible to external influences.

The cavity, first described (v. Ebner, 1897) in the taste-buds of the tongue, is generally flask-shaped (fig. 1 and Plate II. A), and has its mouth directly opposed to the taste pore. It extends but a short distance into the upper segment of the bud, and is formed by the approximation of the peripheral ends of the supporting cells. Projecting into it are the peripheral ends of the taste-cells which pass between the supporting cells. The contents of this cavity are unstainable; it probably contains, as in the tongue (Poirier et Charpy, 1902), a clear liquid, in which the terminal points of the gustatory cells are bathed.

Shrinkage of the bud from its epithelial capsule through the action of a fixing agent is more common than in the taste-buds of the tongue of the same animal. It is so constant a feature that one may well enquire whether the protoplasm of the bud is not more capable of reacting to fixatives than is the protoplasm of the surrounding cells.

From the stability of the capsule, from the minuteness of the pore, from the relative density of the subepithelial tissue at the insertion of the bud, from the ease with which shrinkage occurs, the impression is given that the bud is an organ subject to very little external pressure, and that the cells of which it is composed are capable of reacting freely to any agent which may influence them at their exposed parts in the central flask-shaped cavity of the bud.

The anatomical investigations of the relation of nerve-endings to taste-buds have been made on those of the tongue, with the exception of some work done by Arnstein (1897) on those of the larynx. There is still much doubt as to how the nerves end. It is generally admitted that there is a subgemmal plexus (Retzius, 1892), but as to the nerves that form it and how its constituent elements are distributed, we have as yet no precise information. Thus Jacques (1894) derives perigemmal, intragemmal and intergemmal fibres from a subgemmal plexus, which can thus bear no similarity to the subgemmal plexus or "cupula" of v. Lenhossék (1893) and Arnstein (1897), still less to the plexus of special sensory fibres described by Dogiel (1897). Moreover, no sufficient evidence has been advanced for the view that certain nerves going to the bud are sensory, while others are nerves of special sensation. Again, though Arnstein (1893) appeared to have clearly shown that Fusari (1891) was mistaken in describing the actual connection of the nerve-fibre with the fusiform cell, this connection appears to be still maintained by some authors (Finocchiaro, 1904). Lastly, there is the question of the presence of a ganglion cell in the subgemmal plexus, which Jacques (1894) supports, and Roeske (1897) denies.

I find that the mode of ending of the nerves in the taste-buds of the larynx is as complex as that of the tongue. Lying under the epithelium of the laryngeal surface of the epiglottis and aryteno-epiglottic fold there is a subepithelial plexus composed of both medullated and non-medullated fibres. From this plexus there go to the taste-buds not less than two, and usually three or more, nerve-fibres, some fine varicose fibres, others, which are at first medullated, but lose their sheath at a varying distance between the plexus and the bud. These fibres divide repeatedly, so that at the base of the bud the nerves are very numerous and individual fibres are often difficult to follow (Plate II. A). But two distinct groups can, as a rule, be distinguished: (1) Those that break up to form a plexus at the base of the bud, with prolongations within the bud; and (2) those which go more or less directly round the bud. The latter may branch in the plexus, but there is no anastomosing nor interlacing of branches, as occurs with the fibres of the plexus. In this complex arrangement three groups can therefore be distinguished:—

(I.) Subgemmal plexus; (II.) Intragemmal nerve-fibres; (III.) Perigemmal nerve-fibres.

The so-called intergemmal fibres (Jacques, 1894) are nerves distributed to the epithelial cells which lie between the buds (Plate II. A). They have no direct connection with the bud, and differ in no way from the nerves to the adjacent epithelium.

(I.) The subgemmal plexus Plate III. B, and Plate II. A) lies in the subgemmal connective tissue, and may begin at some distance beneath the bud. The apex is central towards the subepithelial plexus; the base is broad, and applied to the convex central end of the taste-bud. It is formed by branching and apparent anastomosis of one or more fibres which come from the subepithelial plexus. The winding and interlacing of these fibres are very complex, and often are made more difficult to follow because of the "Endbäumchen," which branch around and among them, but from which they are distinct. The plexus is continued within the bud, so that on cross-section the basal part of the cells of the bud appears to be embedded in the plexus.

(II.) There are two sources from which intragemmal fibres arise: (1) From the subgemmal plexus; (2) directly from the subepithelial plexus.

(1) The fibres connected with the subgemmal plexus are very irregular in their course. At the base of the bud they have a plexiform arrangement, and surround the bud cells. Two varieties of endings are seen in connection with this group (Plate II. E): (a) A branching and interlacing network around the cells. This is well marked in the lower third of the bud, less in the middle, and only slightly at the upper third. (b) Knob-like endings most marked at the upper third of the bud; they lie on the cells.

(2) A few non-medullated fibres come directly from the subepithelial plexus and take no part in the formation of the subgemmal plexus (Plate II. B, and Plate III. A, where a fibre is marked v.). They may run to the bud outside the subgemmal plexus, or they may pass through it. In the latter case they run a course more or less direct towards the bud; they may divide in the plexus, and their rami run in opposite directions, but in no case do they show such complex interlacing as do the fibres of the plexus. Their course to the bud is by no means difficult to follow. Within the bud they are not always easy to follow, but the general trend of all the branches is towards the apex of the bud.

(III.) The perigemmal nerve-fibres (Plate II. C and A) form a plexus of non-medullated varicose fibrils which lie around the bud, forming at the apex a circle around the pore (Lenhossék, 1893). They arise from medullated nerves which come from the subepithelial plexus, but which lose this sheath before reaching the base of the bud. They may pass through and even branch in the subgemmal plexus. On cross-section branches may at times be seen to pass from the perigemmal plexus within the bud.

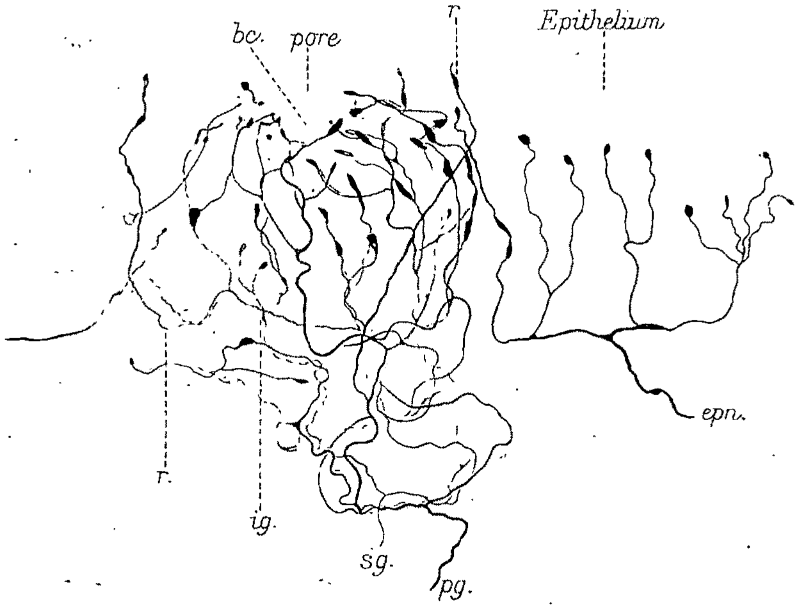
That some of these fibres are sensory as distinct from nerves of special sensation is demonstrated by the following; if we assume that one nerve-fibre carries a special sensation, or if we assume that the special sense organ of taste must have, like the eye or ear, special sensory fibres: (*a*) There can frequently be seen a nerve-fibre dividing into two trunks, one of which goes around or within the bud, the other to the surrounding epithelial cells (Plate II. A). (*b*) Occasionally, and more often near the base of the epiglottis and in the aryteno-epiglottic fold, a division at a node may send one fibre to the bud, another to an unencapsulated nerve-ending, as, for instance, a subepithelial end-tree (Plate III. A). The general course of these fibres is to surround the bud. Their terminals are directed to the apex of the bud to end near the bud cavity.

There are other fibres independent of these, different in structure and mode of distribution, which lie principally at the basal part of the bud and which form the subgemmal plexus and its continuation with the bud. These we regard as nerves of special sensation.

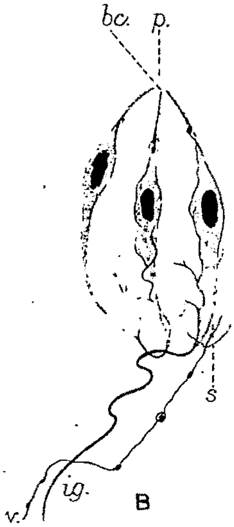
Ganglion cells have been described in connection with the nerves of the taste-bud by many investigators—Retzius, v. Lenhossék, Jacques, &c. Lenhossék (1893) gives a very guarded opinion: "Vollentwickelte Nervenzellen, wie etwa die des Sympathicus, sind es gewiss nicht" (p. 124). In the subgemmal plexus I have never seen sufficient evidence, in vital injection preparations, to enable the deduction to be drawn that swellings which may be seen on the nerves represent ganglion cells. Nor am I convinced of their presence by the evidence afforded by such swellings as are found in Golgi preparations.

The whole question of swellings in the course of the non-medullated nerve trunks is an interesting one, and many varieties are present in connection with the nerves of the taste-bud. For example, there are the well-known varico-

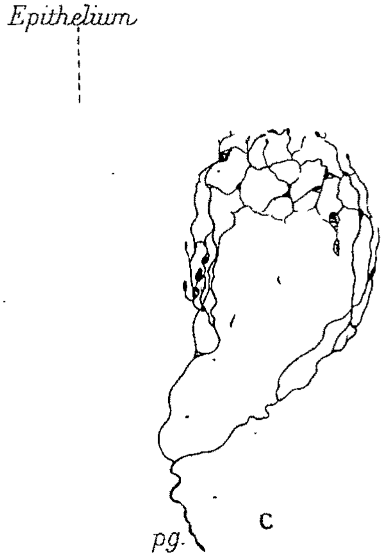
Plate II.



A



B

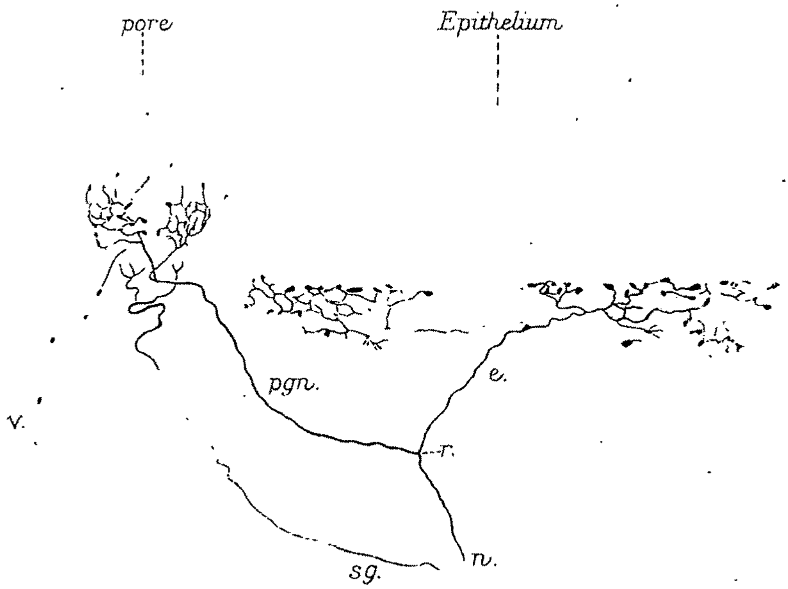


C

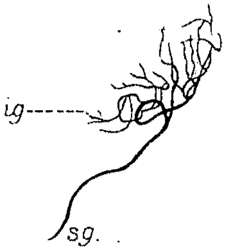
L. H. W. del

Bale & Danielsson, Ltd lith.

Plate III.



A



B

L. H W. del.

Bale & Danielsson, Ltd lith

sities on the fine non-medullated nerve-fibres so constant in their presence and in their form both in non-fixed and fixed preparations that they cannot be passed over as merely *artefacts* or *post-mortem* changes (Kalius, 1905), but must be regarded as representing some peculiarity of the nerve-fibre. Thus it may represent a swelling on the axis cylinder or a point where the fibre is more exposed to external influences, just as at the node of a medullated nerve, where the sheaths are interrupted, there is a tendency to a darker and more profuse staining. Another variety of swelling appears at the point of division of an axis cylinder, due to a diffuse staining where its constituent parts are separating. This swelling will be obviously accentuated if, not two fibres branch off, as is usually the case, but three, four or five, as may at times be seen in the fibre going to the bud or to an adjacent tree ending. There is also that form which occurs at the interlacing of fibres. In addition to these there are a number of swellings of many forms and sizes—oval, triangular, &c. Many of them are artefacts, but whether they can all be classed as such may be open to doubt. This, however, may be said: they are fewer in well-stained preparations and are larger and more numerous in nerves undergoing degeneration.

From the time Verson (1872) described the taste-buds of the larynx various opinions have been held in regard to their function. Verson held that the very fact that they are found in the posterior surface of the epiglottis is proof enough that they are not taste-buds. Krause believed that they were the organs "für intensive Nachgeschmacke."

That taste can be perceived in the upper part of the larynx cannot be doubted. My own investigations corroborate the statements of Michelson (1891) and Kiesow (1901-2) on this point. By means of the laryngoscopic mirror one can touch any desired point of the vestibulum laryngis. Taste perception is rapid and sufficiently accurate to differentiate more or less definitely the four primary tastes, though quantitatively and qualitatively inferior to the tongue.

The following hypotheses have been advanced to explain the presence of taste-buds in the larynx:—

(1) They are a phylogenetic residue.

(2) They are organs of taste whose chief function is to strengthen the reflexes which close the laryngeal cavity during the passage of food: "Die Reflexe zu verstärken wenn Geschmackstoffe in den Larynx gelangen" (Kiesow, 1901).

The first hypothesis is supported by: (*a*) The history of the taste-buds in the vertebrates—a history which shows a formerly widely diffused sense-organ becoming more restricted in locality; (*b*) the history of the taste-buds in the individual life of man, since in early life they cover a much wider area; (*c*) the statement that they can serve no function, as they never come into actual contact with food.

It must be acknowledged that the taste-buds have undergone a phylogenetic decrease, and that in the individual life of man their area of distribution diminishes. We have a corresponding phylogenetic decrease and restriction of area in the allied sense-organs of smell (Mann, 1905).

But restriction of area need not indicate a phylogenetic residue, and may equally well indicate a more specialised sense arising from a concentration of the area of peripheral sense. It is hard to believe that these organs have no function; their number, their abundant nerve supply, their persistence during life, their physiological activity, speak against the acceptance of this hypothesis. One feels that the reason for their continued presence here has to be sought in the intricate mechanism of deglutition.

In discussing the second hypothesis it is well to recall how great a part of the function of the larynx lies in protecting the entrance to the trachea (Anderson Stuart, 1892), and to note that the hypothesis which accounts for the closure of the larynx during deglutition as due to the mechanical lid-like action of the epiglottis cannot be upheld, and that the evidence clearly shows that closure of the aditus laryngis occurs by an approximation of the arytenoids, by contraction of the soft parts together with their apposition to the inferior surface of the epiglottis (Anderson Stuart, 1892; Eykman, 1904). The primary purpose of taste was probably to select a suitable food supply, and so to act as a protective agent for the animal. What more suitable end-organ to assist in protecting the trachea during the passage of food could there be than one whose natural stimulus is the food? In the vestibule of the larynx those functionally active buds, particularly rich in their nerve supply, rest on an exposed surface in marked contrast to the concealed nature of these organs in the tongue; there is, therefore, a rapidity and facility of reaction of laryngeal buds in contrast to the persistence of functional activity apparent in those of the tongue. To this hypothesis the localisation of these organs lends additional support. They are not, as a rule, found at the apex; they become apparent near the middle and are most marked, very obviously so in the dog, on both sides of the line joining the aryteno-epiglottic fold. In man, so far as our preliminary investigations have shown, there are none at the apex of the epiglottis, but they lie round and beneath the cushion of the epiglottis. They are numerous on the medial surface of the arytenoid cartilage and on the lateral surface close to the apex. (Kalius, 1905.)

Further investigation will have to be made, especially as to whether or not a difference in intensity of closure of the larynx results from the application of sapid or non-sapid bodies; but even from the above one may well be justified in claiming some support for the hypothesis that these end-organs act as sentinels to assist in the protection of the laryngeal cavity during the passage of food.

BIBLIOGRAPHY.

ARNSTEIN, C. 1893. "Die Nervenendigungen in den Schmeckbeckern der Säuger." *Arch. f. Mik. Anat.*, Bd. xli., S. 195.

ARNSTEIN, C. 1897. "Die Nervenendigungen und Ganglien der Respirationsorgane." *Anat. Anzeiger*, Bd. xiii., S. 12.

DOGIEL, A. S. 1897. "Ueber die Nervenendigungen in den Geschmacksendknospen der Ganoiden." *Arch. f. Mikr. Anat. u. Entw.*, Bd. xlix., S. 769.

Dogiel, A. S. 1903. "Ueber die Nervenendapparate in der Haut des Menschen." *Zeitschrift f. Wissenschaftliche Zoologie*, Bd. lxxv., S. 46.

Ebner, V. von. 1897. "Ueber die Spitzen der Geschmacksknospen." *Sitz-Ber. Akad.*, Wien., Bd. cvi., Ac. iii., S. 73.

Ehrlich, P. 1902. "Ueber die Beziehungen von chemischer Constitution, Vertheilung und pharmakologischer Wirkung." *v. Leyden-Festschrift*, S. 15.

Eykman, P. H. 1903. "Der Schlingact dargestellt nach Bewegungsphotographien mittelst Röntgen-Strahlen." *Arch. f. d. ges. Physiol.*, Bd. xcix., S. 513.

Finocchiaro, G. 1904. "Contributi allo Studio delle terminazioni Nervose nelle papille circumvallate." *Arch. Ital. d. Anat. e. d. Embriologia*, v. iii., p. 289.

Fusari et Panasci. 1891. "Les terminaisons des nerves dans la muqueuse et dans les glands sereuses de la langue des mammifères." *Arch. ital. de biolog.*, T. xiv., p. 240.

Gräberg, J. 1899. "Zur Kenntnis des cellulären Baues der Geschmacksknospen des Menschen." *Anat. Hefte*, Bd. xii., Hft. 2, S. 339.

Jacques, P. 1894. "Terminaisons nerveuses dans l'organe de la gustation." *Thèse de Nancy.*

Kalius, E. 1905. "Sinnesorgane." *Handbuch der Anat. des Menschen. von K. v. Bardeleben*, Bd. v., Ab. 1, S. 243.

Kiesow, F. 1902. "Sur la présence de boutons gustatifs à la surface linguale de l'épiglotte humaine, avec quelques réflections sur les mêmes organes qui se trouvent dans la muqueuse du larynx." *Arch. ital. de biol.*, T. xxxviii., p. 334.

Kiesow, F. 1901. "Ueber Geschmacksempfindungen im Kehlkopf." *Arch. ital. de biol.*, T. xxxvi., p. 94.

Krause, W. 1870. "Die Nervenendigungen der Zunge des Menschen." *Göttinger Nachrichten*, No. 21, S. 424.

Lenhossék, M. v. 1893. "Der feinere Bau und die Nervenendigungen der Geschmacksknospen." *Anat. Anzeig.*, B. viii., S. 121.

Mann, G. 1905. "On the Thalamus." *Brit. Med. Journ.*, Feb. 11, p. 290.

Michelson, P. 1891. "Ueber das Vorhandensein von Geschmacksempfindung im Kehlkopf." *Arch. f. Path. Anat.*, Bd. 123, S. 389.

Poirier et Charpy. 1901. *Traité d'Anatomie Humaine.* T. iv., Fas. 1, p. 132.

Ranvier. 1882. *Traité technique d'histologie;* quoted by Poirier et Charpy. *Anat. Humaine*, T. iv., Fas. 1, p. 133.

Retzius. 1892. "Die Nervenendigungen in den Geschmacksorganen." *Biol. Untersuch*, iv.

Roeske. 1897. "Ueber die Nervenendigungen in den Papillæ fungiformes der Kaninchenzunge." Berlin. (Inaugural thesis.)

Shofield, R. H. A. 1876. "Observations on Taste-goblets on the Epiglottis of the Dog and Cat." *J. of Anat. and Physiol.*, vol. x., p. 475.

Stuart, T. P. Anderson. 1892. "On the Mechanism of the Closure of the Larynx." *Proceed. of the Royal Society of London*, vol. l., p. 323.

Verson. 1871. *Strickers Handbuch der Lehre von den Geweben des Menschen und der Thiere.* Bd. i., S. 456.

Wilson, J. Gordon. 1904. "The Relation of the Motor Endings on the Muscle of the Frog to Neighbouring Structures." *J. Comp. Neurol. and Psychol.*, vol. xiv., p. 1.

Explanation of Plates II. and III.

Plate II. a.

Section of the epiglottis of a rabbit. Taste-bud lying in a shallow depression of the epithelium with pore (*p*) communicating with the faintly outlined cavity (*bc*) in the upper segment of the bud. (*pg*) Nerve dividing in submucous tissue and finally breaking up into numerous branches, which form a network around the bud (perigemmal plexus); branches (*r*) are seen passing into the surrounding epithelium. (*sg*) Nerve-fibre branching frequently to form a network (only partly shown) at the base of the bud; it sends branches (*ig*) within the bud (intragemmal fibres). (*epn*) Nerve with epithelial endings.
Zeiss D. Comp. occ. 4.

Plate II. b and c.

Section from mucous membrane of vestibulum laryngis of dog, near base of epiglottis. (B) Bud with spindle-shaped cells (*s*) stained by methylene blue, showing relation of the intragemmal fibres to the cells. (*v*) Fine varicose fibre passing into bud. (C) Bud cut obliquely with the part adjacent to the pore not seen. The perigemmal plexus is connected with the nerve (*pg*). At the base a few bud cells are seen in outline.
Zeiss $\frac{1}{12}$ *oil immersion. Comp. occ.* 4.

Plate III. a.

Mucous membrane from larynx of dog near arytenoid cartilage. (*n*) Nerve-fibre which divides at (*r*) into two branches, of which one (*e*) goes to end in a subepithelial end-tree (Endbäumchen), the other (*pgn*) goes to form a plexus around the bud. The nerve (*sg*) winds spirally and branches at base; it finally sends off shoots (not shown) into bud. (*v*) Fine varicose nerve-fibre passing into the bud.
Zeiss D. Comp. occ. 4.

Plate III. b.

From epiglottis of a dog; lower segment of bud which has been cut obliquely. The nerve (*sg*) coils and branches in subgemmal connective tissue to form a plexus around the base of the bud (subgemmal plexus). From this plexus branches (*ig*) pass into the bud (intragemmal).
Zeiss $\frac{1}{12}$. *Comp. occ.* 4.

I am indebted to Mr. L. H. Wilder for the carefulness and accuracy with which he has prepared these drawings.

Review.

Mental Defectives. By MARTIN W. BARR, M.D. London, Rebman Limited, 1904, pp. 368.

Dr. Barr, who is chief physician to the Training School for Feeble-minded Children at the well-known institution at Elwyn, Pennsylvania, is well qualified by his position for writing a book on this important subject. He commences by giving the synonyms of idiocy, imbecility, &c., in different languages, and alludes to the definitions which have been applied by many authorities to this class of cases. In America, the term feeble-minded has been used to cover every form of mental defect, and it is well that this should be known, because in England this term "feeble-minded" has been used especially for cases above that of idiocy and imbecility, and it is children belonging to this feeble-minded class who go to the special classes and schools which have been established for them in London and different parts of the country. In fact, the Elementary Education (Defective and Epileptic Children) Act, 1899, expressly excludes imbeciles from these classes, as the imbeciles have insitutions built for them and an Act, the Idiots Act, 1886, under which these institutions work. Fifty-four pages are devoted to the history of the way in which different countries have taken up the work, and the education of the savage boy of the Aveyron by Ibard is fully described. The classifications of Esquirol, Duncan and Millard, Langdon-Down, Ireland, Shuttleworth, and Kerlin, who preceded him, are alluded to, and the author adds another one which has a chiefly educational foundation as distinguished from a pathological one. In the chapter on etiology the 2,380 cases collected by Fletcher Beach and Shuttleworth are mentioned, and the author compares with these 3,050 cases which he has collected from different sources. There are some differences, however: thus phthisis accounts for 28·31 cases in the English tables, but for only 7·57 in the American. Again, insanity and imbecility produce 21·38 in the English tables, but only 7·08 in the American. Epilepsy and other neuroses account for 20·00 in England, but only 5·58 in America. Intemperance, which produced 16·38 in England, only produced

4·46 in America, which seemingly would go to prove that the American race is more temperate than the English. However, causes acting before birth account for 64·85 cases, those at birth for 2·92, and those acting after birth to 32·23, and allowing for differences in numbers much the same ratio prevails in the English tables. The chapters on diagnosis, and training and treatment are excellent. In America, public opinion apparently is not so markedly against asexualisation as in England. The prevailing opinion in enlightened circles here is that hopeless cases should be shut up for life, but in America they would not only do that, but asexualise them as well. Dr. Pilcher, it appears, castrated fifty-eight boys, and in almost every case there was marked improvement, both mental and physical. The author's preference would be for oöphorectomy in the female and testiectomy in the male. Sixty-five pages are devoted to illustrative cases, and thirteen to the responsibility or irresponsibility of Samuel Henderson, a murderer. There is a copious bibliography at the end of the book, and there are a large number of photographs of cases, but in another edition these should be better arranged. For instance, when one is reading about epilepsy one does not wish to see photographs of cases of cretinism and myxœdema, and when reading the psychology of "idiots savants," photographs of microcephalus do not help one much. After all these are minor defects, and if anxious parents and earnest teachers for whom the author says the book is written will read, mark, and inwardly digest it, nothing but good can result.

FLETCHER BEACH.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

At the Meeting held at the National Hospital, Queen Square, on Saturday, June 24, at 10 a.m., the following cases were shown:—

Dr. Beevor.—(*a*) *Progressive muscular atrophy, immobile pupils, exostosis on humerus.*

[12] A. R., male, aged 38, French polisher. Onset four years ago with cramps in fingers, wasting of thenar eminence and first interosseal space, forearm, and, lastly, biceps. No pain, numbness, nor loss of feeling. Disease seems to have progressed more rapidly in past six months. Gonorrhœa ten years ago. No history of lues. Married; no children; no miscarriages.

Present Condition.—Fairly well nourished. Bony exostosis under tendon of left biceps. Smaller one on right humerus. Pupils—pin-point, R. > L. Argyll-Robertson—Do not dilate to shade, dilate slightly to cocaine, vision $\frac{6}{6}$ R. and L. Symmetrical atrophy slight in triceps; marked in biceps, extensors, thenar eminence, abductor indicis and interossei. Weakness of corresponding movements.

Electrical reaction—Partial R. D. in atrophied muscles. Small muscles of thumb do not react to faradism or galvanism.

Reflexes.—Triceps, knee-jerks and ankle-jerks normal. Biceps and supinator-jerks absent. Plantars flexor.

(*b*) *Peroneal type muscular atrophy—familial type.*

[13] J. T. S., male, aged 38, tailor. Twenty-one years ago, difficulty in walking. Wasting of calf muscles followed by wasting of thenar eminence. Wasting has progressed more rapidly in last two years. No pain, tingling, nor loss of sensation. One brother and one sister similarly affected; both younger than patient. Brother is well advanced. Sister affected only in one foot. In neither are the hands affected.

Present condition.—Some wasting of forearms. Atrophy of small thumb muscles. Dorsal and palmar interossei. Weakness in corresponding movements. Some fibrillary twitchings in small muscles of thumb. Atrophy of calf muscles and peroneal group, pes equino varus. Weakness in corresponding movements of feet. Electrical examination shows change in or absence of reaction of muscles to faradism and galvanism.

Reflexes.—Triceps, biceps, supinator, and knee-jerks normal. Ankle-jerk absent. Plantar flexion, epigastric, abdominal, and cremasteric, normal.

Gait.—Can scarcely stand without mechanical support. Has had several operations of tendo-Achillis and plantar fascia performed by Mr. Fisher.

(*c*) *Mesencephalic lesion.*

[14] M. J., female, married, age 39. Eleven years ago staggered in walking. Seven years ago difficulty in speech and could not close right eye nor move either eye out. Five years ago stiffness in right arm, with ataxia. Two years ago regurgitation on swallowing. One year ago micturition difficult, numbness of fingers, diplopia. Eight months ago vision diminished.

On admission.—Slow, deliberate, staccato, syllabic speech, somewhat slurring, accompanied by side to side tremor of head; laughter explosive; visual field restricted to 5°. Post-neuritic atrophy.

Lateral conjugate deviation impossible; on attempt convergence of both. Upward and downward movement present; pupils equal and regular, react to light and accommodation. Paralysis of right side of face. Watch heard only when applied to ear.

Pharyngeal reflex absent, deglutition difficult, speech as above. Soft palate moves weakly, tongue protruded normally, inco-ordination and intention tremor.

Lower extremities rigid and inco-ordinate. Complete anæsthesia, analgesia; loss of sense of position, astereognosis; diminution in weight sense.

Reflexes:—Knee-jerks active, plantars right flexion, left (?) flexion; abdominal and epigastric slight.

Dr. J. H. BRYANT.—(*a*) *Two cases of the peroneal type of family amyotrophy.*

[15] *Case* 1.—Lilian S., aged 12, first came under observation in April, 1901, for wasting of the muscles of the legs and hands.

Forceps were used at her birth, but there was no apparent injury to her brain. She began to walk at the age of 10 months. When 4 she had an attack of whooping cough, after which she was unable to walk properly. On account of this trouble she was taken to an Orthopædic Hospital, where a diagnosis of infantile paralysis was made, and she was ordered to wear boots with iron supports.

At this time both feet and legs were wasted up to the knees. When 5½ she had an attack of scarlet fever and at 6 and 8 attacks of measles. In January, 1900, wasting of the muscles of the hands was noticed.

Both her parents are strong and healthy. A paternal uncle is suffering from a similar disease characterised by weakness and wasting of the legs and hands. A brother (Case 2) is also affected with a similar disease. She has two brothers aged 18 and 17, who are quite well.

Condition.—The feet, hands and legs are weak. The gait is somewhat "high-stepping" in character. There is slight talipes varus.

There does not appear to be any loss of sensation to touch, pain, heat or cold, but it is curious that strong electrical stimuli applied to the hands cause no feeling of discomfort or pain.

There is marked wasting of the thenar, hypothenar and interossei muscles of both hands.

The muscles of the legs are much wasted.

There is no wasting of the deltoids, scapular or facial muscles. Slight fibrillation has been noticed at times in some of the muscles. There are no hypertrophied muscles.

The knee-jerks cannot be obtained. The plantar reflex is present, and is flexor in character. Neither wrist- nor elbow-jerks can be elicited.

On stimulation, both with the faradic and the galvanic currents there is no response in the thenar, hypothenar, and interosse muscles, the small muscles of the feet, the peronei, tibialis anticus and extensor longus hallucis.

[16] (*b*) *Case* 2.—Percy S., aged 8, first came under observation in August, 1903. Forceps were also used at his birth, but there was no apparent injury to the brain. He was able to walk and run about as other children until two and a half years ago, when he had an attack of measles, after which his mother noticed he was weak on his legs and could not lift his big toes. He seemed to be helpless, and began to fall down frequently on account of his legs giving way under him. When first seen,

the paralysis of the extensor longus hallucis muscles was the most striking feature.

He has become much worse, and now cannot walk at all by himself unless his legs are supported by straight internal splints extending from the foot to just below the knee.

The feet are dropped, the big toes are flexed, and the feet are in a condition of talipes equino varus and pes cavus.

The thenar and hypothenar muscles have commenced to waste.

An attempt was made to test the electrical reactions, but he was so frightened, and struggled so violently, that it was impossible to obtain any reliable data.

Dr. George Ogilvie.—*A case of tabetic gait beginning at the age of* 64.

[17] Male, aged 73, formerly a storekeeper. He was in good health up to the age of 64; since then progressive weakness and unsteadiness of the legs. Unable to walk alone during the last five years.

Present state.—Mental condition good. Speech slow and hesitating.

Lower limbs.—Strength poor, but no wasting. Cannot walk or stand alone. Tabetic gait. Knee-jerks absent. Much sweating of the legs and feet. Skin poorly nourished. No lightning pains or cramps. No anæsthesia. Muscular sense good.

Upper limbs.—Strength poor. Grip = $\frac{1}{4}$ normal. Slight tremor and inco-ordination.

Sight.—Becoming worse during last year. R. $\frac{6}{12}$, L. $\frac{6}{18}$. Pupils react to light and accommodation. Nystagmoid movements on extreme excursion. Slight general contraction of the fields of vision. Lenticular opacities. Subsiding optic neuritis, passing into atrophy on the left side.

No girdle sensation. No visceral crises. No incontinence.

Dr. H. Campbell Thomson.—*Case of tremor.*

[18] M. S., female, aged 34.

Fifteen months ago suffered considerable shock owing to the death of a relative, and soon after she noticed the left arm began to tremble.

There is now a rhythmical tremor of left arm, varying in intensity, but said to be never absent, except during sleep. It is usually rather worse during voluntary movement. General health is good.

Dr. T. D. SAVILL.—*A case of ataxy in a woman, aged* 40, *of twenty years' duration.*

[19] Margaret C., aged 40, has been unable to walk properly for twenty years. The difficulty came on quite suddenly sixteen days after a severe fright, consisting of her mistaken apprehension by the police. According to her own account, the difficulty of movement was almost as great then as it is now, and it has not varied much, excepting when she had "Viters's dance" thirteen years ago. There is no definite history of syphilis.

At the present time the symptoms are mainly two in number, *inco-ordination* of all the limbs, sufficient in the legs to prevent her walking or standing without crutches, unaccompanied by paralysis or atrophy, and increase of all the *tendon reflexes*, Babinski's reflex also being present. The speech has an explosive or staccato quality, suggestive of disseminated sclerosis, and the teeth are the seat of pyorrhœa alveoloris. The pupils, fundi, and fields of vision (tested by Mr. J. L. Tomlinson) are normal, and there is no nystagmus, no sensory or sensorial changes. Strong faradisation for two or three weeks has had no notable effect; suggestions as to treatment are solicited.

Dr. LEES and Mr. BALLANCE.—*Posterior-basic meningitis in a boy of* 3 *years, resulting in hydrocephalus, emaciation, and a comatose condition which had lasted for three weeks, during which he had not recognised his mother, and had required nasal feeding. Trephining: drainage of lateral ventricle by rubber tube into subarachnoid space for twenty-two days; cerebral hernia subsequently protected by insertion of a platinum plate. Complete recovery except for deaf-mutism.*

[20] The operation was performed by Mr. Ballance seven years ago (1898). The boy has remained in good health, and is being educated in a special school for deaf mutes. He is now 10 years old.

This is the successful case mentioned in the concluding words of the paper on "Simple Meningitis in Children," by Dr. Lees and Sir T. Barlow, in the seventh volume of Allbutt's "System." The case has not previously been exhibited at any Medical Society.

Dr. THOMAS BUZZARD.—(*a*) *Case of mesencephalic lesion.*

[21] E. P., aged 35, spinster. Suffered from fatigue seven years ago. Same time loss of vision. Tremor four years. Five years diplopia. Dimness of vision increased during past year. No history of headache or vomiting. No sphincter trouble.

Present condition.—Discs pale and atrophic. Pigmentation of choroid. Complete ophthalmoplegia interna and externa. Pupils react to light, but not on accommodation. Teeth show well-marked trophic changes. Slight weakness of left face.

Gait.—Tends to deviate to right. Well-marked tremor on movement confined to right side. Reflexes, sensory system and muscular system normal.

(*b*) *Case of muscular wasting with unusual symptoms.*

[22] H. C., aged 45. June, 1904, aching pain right leg. September, 1904, right foot began to drop. Later weakness of right knee. January, 1905, burning pain in left foot. March, 1905, weakness of left leg. Thighs have wasted since November, 1904. Same time fibrillation of thighs and buttocks noted.

Present condition.—Congenital nystagmus. Marked fibrillation of upper extremity.

Lower extremity.—All movements of toes and ankles absent. Very slight flexion and extension of knee. Slight extension of thigh. All other movements absent. Muscles below knee do not react to faradism. React briskly to galvanism. No polar change. Ankle-jerks and plantar responses absent. All other reflexes present and normal. Sensory system normal.

Dr. F. E. BATTEN.—(*a*) *Congenital affection of sixth, seventh and twelfth cranial nuclei.*

[23] E. S., aged 9. First seen when 2½ years old, the condition having remained unaltered since that time.

The child has had facial paralysis since birth, and was never able to suck.

First child. Father and mother healthy.

Present condition.—Intelligent child, with partial paralysis of both sides of the face, the right being more affected than the left.

Complete paralysis of lateral movements of the eyes.

Convergence good. Pupils equal. React well.

Atrophy of the tongue, both sides.

Knee-jerks active. Plantars flexor.

(*b*) *Case of tremor of right arm associated with epilepsy.*

[24] R. C., girl, aged 20. Twitching of right hand first noticed in April, 1904, and shortly after this patient had a fit in which she fell, but did not bite her tongue. No headache or vomiting.

The girl has quick, sudden contractions of the muscles of the right arm, which became more marked on intention movement. The power of the hand is good.

There is no sign of organic disease. The plantar reflexes are difficult to obtain. Epileptic fits occur at irregular periods—but are controlled by bromides. No improvement has taken place in the condition during the past twelve months.

(c) *Case of acute ataxia in a boy.*

[25] The boy, aged 3½, had measles in March, 1904, and there was a family history of tuberculosis. Upon being got up he became dazed and then unconscious, and remained so for a week. He then had convulsions and tremors, and on regaining consciousness he could not sit up nor speak. A month later he had marked inco-ordination of both legs and arms. There was no paralysis. The knee-jerks were well marked, with a tendency to ankle clonus. Plantars generally flexor. He swallowed well, and spoke in a low, hesitating manner. He has complained of pins and needles in his limbs. His mother complains that since his illness his moral sense has become much perverted. He was seen again in May, 1905, and marked improvement had taken place in his gait.

Knee-jerks still active. Plantars flexor.

Dr. A. F. VOELCKER.—(a) *Case of acute ataxy.*

[26] D. F., aged 4¾ years, female.

This child was shown at a meeting of the Neurological Society on March 30, 1905, as an example of acute ataxy.

She was supposed to have had an attack of scarlet fever in Christmas, 1904, in which she became unconscious for fourteen days; gradually regained consciousness, but has been unable to stand or feed herself since. Her speech was lost, but has gradually been returning. No vomiting during the initial illness.

On admission the child was unable to walk or even stand, and could hardly sit up alone in bed. Marked tremor of the head. When supported, her gait is very markedly ataxic; the toes are pointed down. At times progression is cross-legged. Some ataxy of upper limbs. Some tendency to fall backwards.

Marked muscular hypotonus. Plantar reflexes variable, frequently extensor on the left side. Knee-jerks brisk; no A.C. Speech slow, drawling, indistinct.

During May the child has improved somewhat, and makes very little effort to walk.

The knee-jerks are less readily obtained. The ataxy of both lower and upper limbs is less, and the mental condition is improving.

(*b*) *Case of* (*?*) *disseminated sclerosis.*

[27] M. C., aged 11, female. In June, 1903, she was noticed to be losing her power of walking, and to become weak in the arms and legs, and to have difficulty with her speech. This has been getting gradually worse.

Since January, 1905, she has been unable to walk or stand, and has been unable to feed herself. The impairment of speech has not become more marked. There has been no vomiting, and only an occasional headache. She has, however, complained occasionally of pain in the lower part of the back and in the back of the neck. She has occasional attacks of vacancy.

Well nourished, skin pigmented. Upper central incisor notched. Spleen felt 1½ ins. below the costal margin. No scars or other evidence of congenital syphilis.

Nervous system.—Fairly intelligent. At times emotional, at others apathetic.

Speech.—Slow, drawling.

Motor.—Can move all her limbs. Grip good. No muscular wasting. Marked intention tremor, especially in upper limbs.

Gait.—Very unsteady. Can only stand and walk with support. Gait ataxic, but feet plantar in fairly regular series of positions. No cross-legged progression. Marked tremor of head when patient drinks. Voluntary movements increase the tremor of the upper limb. No tremors during repose.

Sensory.—Beyond a slightly prolonged latent period nothing abnormal detected.

Cranial nerves.—No optic neuritis or atrophy. No nystagmus. Pupils react to light and accommodation. No palate palsy.

Reflexes.—Plantars variable, more often flexor than extensor, sometimes not obtained.

Knee-jerks distinctly increased. No patellar clonus. No true ankle clonus. Reflexes of upper limb slightly exaggerated. Jaw-jerk not exaggerated. Sphincters natural.

Dr. H. Lewis Jones.—(*a*) *A case of paralysis of the upper extremity after a dislocation of the shoulder-joint, in which the distribution of paralysis was unusual.*

[28] The deltoid was almost unaffected and preserved normal reactions, but the biceps, brachialis anticus and supinator longus

were completely paralysed and showed a reaction of degeneration which is still present. Moreover, the clavicular portion of the pectoralis major preserved normal reactions, but the lower (sternal) part showed R. D.

[29] (*b*) *A case of bilateral atrophy of the intrinsic muscles of the hands coming on in a young female patient.*

The wasting has persisted for many years. Weak normal reactions present in the affected muscles.

Dr. Henry Head.—(*a*) *Case of myoclonus.*

[30] W. G., aged 28. Nine years ago began to have "cramp" in the feet and hands. This was soon followed by tremors of the limbs, which have increased in violence, especially recently. The mother's brother is said to be in a similar condition.

He has had no fits. Always has been dull and has never done any work. He can stand and walk with difficulty. The elbows are flexed, forearms pronated, wrists flexed, and fingers extended. The legs are extended and somewhat rigid, the feet are inverted and pes cavus is greater on the right than left side. Coarse bilateral clonic movements at the rate of about 140 a minute are present in both arms, and in a less degree in the lower extremities. These cease during sleep. Sensation is not affected. Both knee-jerks are present, and are equal on both sides. No nystagmus. Discs are normal. The tongue is tremulous. Sphincters are unaffected.

(*b*) *A case of Huntingdon's chorea.*

[31] H. S., aged 38. A little more than twelve months ago he noticed that his legs began to tremble. About seven months ago his speech became affected. He is married and has four healthy children. His brothers and sisters and their children are healthy. No similar condition has appeared in any of his relatives except in his mother's sister who is now 83 years of age. At the age of 40 she is said to have begun to be restless, as he is now, and to have remained so until the present time.

He is a well-developed man, with no disease in the heart or lungs; the urine is unaffected. He speaks like a man who has difficulty in controlling his tongue; each word is begun with some difficulty, and some words are slurred, especially when he is in the presence of strangers. The tongue is extremely unsteady. Face, hands, and legs are in constant restless movement, which is said to cease during sleep. Chest-wall and diaphragm participate in these irregular movements. Tremor is absent. The knee-jerks are normal, and the toes go downwards when the sole of the foot is stimulated. The pupils act normally, and micturition is not affected.

Dr. G. NEWTON PITT.—*Case of Thomsen's disease* (?)

Dr. C. O. HAWTHORNE.—*Case of hemianæsthesia and hemianalgesia, with partial hemiplegia, in a man aged* 34. *Attributed to a blow on the head in November*, 1904.

Dr. LEONARD GUTHRIE.—*Acute ataxy* (*encephalitis cerebelli*).

[32] Patient is a girl, aged 7 years. In February, 1904, at the height of an attack of measles with temperature 103°-104°, she developed cerebral symptoms, vomiting, convulsions, unconsciousness, followed by squint, retraction of head and photophobia. After five days all the acute symptoms subsided, but the patient was aphasic, seemed hyperæsthetic, and rapidly wasted. The optic discs were normal. A month later she was still aphasic, could not sit up or move lower limbs, the knee-jerks were exaggerated, ankle clonus present, but no Babinski plantar reflex was obtained. There were marked inco-ordination of the upper limbs and some athetoid movements of fingers. There was general anæsthesia to pin-pricks, but she recognised hot and cold, and appeared to fear pinching. All limbs were slightly rigid. Sphincter control was lost for two months. Then general improvement occurred. By the end of May, 1904, she could stand with support, feed herself and talk.

Present condition.—Healthy looking, well-nourished child. Slow mentally, but not deficient. Disposition docile and gentle. Speech drawling and bleating in character. Special senses normal. Eyes and pupils normal. Slight ataxy and tremors of upper extremities. She can stand alone and steadily when eyes are closed, but cannot walk more than a few steps without guidance. If left alone she reels, turns half round and falls backwards or to one side or the other. Loss of equilibrium seems due to ataxy in placing the feet rather than to vertigo. Gait is ataxic. Knee-jerks brisk; ankle clonus present at times. Plantar response negative or flexor.

Condition seems due to acute encephalitis affecting chiefly cerebellum or its peduncles.

Dr. WILFRED HARRIS.—*Case of transverse sacral myelitis (first sacral).*

[33] D. K., a Polish Jew, tailor, was seven years ago attacked suddenly by pain across sacral region, and convulsion of both legs for two hours. Since then the legs have always been weak, and his gait much as at present. Incontinence and precipitancy of both sphincters. Sphincter ani reflex good.

P.S.—Weak gait, no foot-drop. Wasting of right gastrocnemius, and especially of the intrinsic foot muscles on both sides. There is well-marked R. D. in the short flexors of toes, and sluggishness of contraction in R. gastrocnemius to faradism.

Knee-jerks normal. Achilles-jerks both absent.

Well-marked anæsthesia on feet and back of calves and buttocks, perinæum, and penis and scrotum, corresponding to the first sacral and lower segments.

Dr. JAMES TAYLOR.—(*a*) *Two cases of achondroplasia.*

(*b*) *Case of tremor of anomalous type in a brass-worker. ? metallic.*

Dr. GOLLA.—(*a*) *Syringomyelia.* (*b*) *Cerebral lesion.*

The afternoon was devoted to a Pathological Demonstration.

Communications by—

Dr. ALEXANDER BRUCE.—"On the Distribution of the Cells in the Intermedio-Lateral Tract in the Dorso-Lumbar Region of the Spinal Cord."

Dr. MOTT.—"On the Brain and Nervous System in Trypanosome Infections, including Sleeping Sickness, and the part played by Diplo-streptococci."

Sir VICTOR HORSLEY and D. R. H. CLARKE.—"On the Relation between the Cortex and Nuclei of the Cerebellum respectively, and the rest of the Nervous System, with Special Reference to a Fresh Method of Investigation."

Specimens Exhibited by—

Dr. LEWIS JONES.—(i.) Twelve photographs of tracings of current waves of medical coils, to illustrate the variety of curves of those currents. (ii.) Induction coils to illustrate the curves of current waves. (iii.) Mechanical interrupters for the production of interrupted currents for diagnostic purposes.

Dr. C. E. BEEVOR.—Injected brains showing the vascular supply of the anterior, middle, and posterior cerebral, and of the anterior choroid and posterior communicating arteries.

Dr. L. HUMPHREY.—The parathyroid glands in Graves' disease.

Dr. W. B. RANSOM.—Syringomyelia with neuro-epithelioma of the spinal cord.

Dr. STANLEY BARNES.—(i.) Healed tubercular meningitis; death from secondary hydrocephalus. (ii.) Sarcomatosis of spinal pia, secondary to cerebral tumour.

Dr. R. T. WILLIAMSON.—(i.) Degeneration in the intramedullary fibres of the spinal posterior nerve roots in tabes dorsalis, diabetes mellitus, and intracranial tumours. (ii.) Changes in the spinal cord associated with severe anæmia.

Dr. E. FARQUHAR BUZZARD.—The morbid anatomy of myasthenia gravis.

Dr. HALE WHITE and Dr. F. A. BAINBRIDGE.—Specimens from case of caisson disease.

Various other specimens from the Museum of the National Hospital were shown.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Society can obtain the Index of "Brain" for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Society the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

[FEBRUARY, 1906.]

BRAIN.

PARTS III. and IV., 1905.

Original Articles and Clinical Cases.

CEREBRAL SCLEROSIS.

BY ALFRED W. CAMPBELL, M.D.EDIN.

From the Pathological Laboratory at Rainhill Asylum,
Lancashire.

With 19 Figures.

TABLE OF CONTENTS.

INTRODUCTION.
CHAPTER 1.—General Remarks on the Neuroglia.
CHAPTER 2.—Development and Growth of the Neuroglia.
CHAPTER 3.—Tuberose Sclerosis.
CHAPTER 4.—Megalocephaly.
CHAPTER 5.—Cerebral Hemiagenesis or Hemisclerosis.
CHAPTER 6.—Lobar Agenesis with Sclerosis and Microgyria.
CHAPTER 7.—Cerebral Arterio-Sclerosis.
CHAPTER 8.—Colloid Sclerosis.
CHAPTER 9.—Giant Cell Sclerosis.
CHAPTER 10.—Other Forms of Sclerosis in General Paralysis of the Insane, Senility, Epilepsy, Idiocy, and Following Cerebral Softening and Syphilis.

INTRODUCTION.

DERIVED from the Greek word σκληρός, the equivalent of the Latin *durus*, the term sclerosis denotes induration. It has been applied, perhaps too freely and with inadequate regard to pathogenesis, to a number of conditions of the brain having increased firmness, local or general, as a common character.

In this paper I propose to consider all the known forms of sclerosis, touching lightly on varieties familiarised to us by frequent occurrence, and giving details of rarer kinds.

Cerebral sclerosis is nearly always a secondary condition, reparatory or hyperplastic, and following some disturbance or destruction of normal constituents. Only in rare instances is it a pure katabiotic product, and even then suspicion of the operation of a primary factor cannot be wholly suppressed.

Viewed from the standpoint of histology, sclerosis is almost invariably synonymous with gliosis; also, it is almost of necessity associated with alterations in the more fundamental constituents of the central nervous apparatus, the nerve cells and their appurtenances, the stations and conductors of nervous impulses. It is seen in its simplest and purest form as an outcome of secondary degeneration of one of the known tracts of medullated nerve fibres, and is then reparatory. It will be well here to glance at the sequence of events which leads up to this particular form of sclerosis. The process begins in necrotic swelling and disintegration of the myelin and possibly of other constituents of the medullated nerve fibres. Then ensue important secondary changes in the nutritive apparatus and supporting tissue of the part. As a mechanical result of the swelling of the fibres, the vascular and lymphatic channels are interfered with and conditions favourable to the emigration of lymphocytes and leucocytes set up; with this is associated an enlargement[1] of the supposed fixed neuroglia residents, brought about by one or both of two causes, defective metabolism from impeded blood and lymph circulation, and irritation by or reaction to the toxic products of myelinic decay (cholin and allied substances). The next stage is one of absorption of the diseased and disintegrated nerve fibres. What the agents are here it is impossible to say, but some would give force to the supposed scavenger function of enlarged neuroglia cells, while others would credit the phagocytic capacity of altered leucocytes and lymphocytes, and probably both are subservient, co-operating in the absorption of diseased products, and in the connection of such products to the lymph and blood channels now relieved of their primary embarrassment. In the final stage the place of the medullated nerve fibres is taken by a poorly vascularised mass of neuroglia or connective tissue cells and their processes, in the midst of which thick-walled vessels, some of them newly formed, ramify. I have seen it stated that cerebral sclerosis is not accompanied by shrinkage, but this does not apply to all forms, and certainly not to the simple form of sclerosis just used as an example. If proof on the point be needed, it is afforded by the alterations in form of the spinal cord seen in cases of tabes dorsalis and in cases of descending degeneration affecting the motor tract.

---

[1] It is a moot point whether the enlarged neuroglia cells we see in such cases are changed residents or formative cells recently derived from the blood-vessels or lymphatics. In my opinion the latter is the case.

The principles seen in this simple instance of the manner in which sclerosis plays a reparatory *rôle*, apply to many varieties of cerebral induration; that which we find so often around old-standing foci of softening and of hæmorrhage, that which is the outcome of impaired nutrition and which affects individual gyri (local microgyria), that resulting from agenesia and affecting whole lobes or even hemispheres, and many other forms, can be grouped as essentially of the same nature. In all an overgrowth of neuroglial constituents is a common character representing Nature's effort to make complementary amends for a deficiency, destructive or developmental, of nerve cells and nerve fibres.

Other forms of sclerosis have a mixed pathogenesis. In causing the initial necrosis of fundamental constituents and the superimposed reparative or repellent proliferation of neuroglia, *an irritant is the primary factor.* This may act locally or generally. The induration observed in general paralysis of the insane, a disease in which we assume for granted the circulation of noxious blood, instances the general condition, the sclerosis round an imprisoned foreign body the local.

Lastly, there are rare forms of sclerosis in which the principal constituent is not neuroglia, but some other element or structure. Thus, I shall have occasion to describe a condition in which hyperplasia of ganglionic nerve cells has been found accountable for the induration, and in this class "colloid sclerosis," with its vascular alteration, must needs be included. These, however, are borderland cases, and it is an open question whether they should not be called outright tumours or new formations.

CHAPTER 1.—GENERAL REMARKS ON THE NEUROGLIA.

Some observations on the neuroglia must be introduced here, because it is the most prominent tissue in nearly all the conditions of which I shall write. Since, however, it is a tissue whose attributes have been discussed at length by many able writers, of whom the late Professor Weigert, von Lenhossek, and Storch, in Germany, and Dr. Ford Robertson in our own country, may be specially mentioned, it is proper that my remarks shall be shorn of detail and, though flavoured with criticism, made pertinent to the histogenesis and pathology of the condition in hand.

First, in order that the tissue may be considered in its relation to sclerosis, we must look at the varieties of neuroglia which have been recognised in the central nervous system.

While others have generally classified varieties of glial elements according to their morphological and structural peculiarities, I have always found it more convenient, paying regard to their chemical affinities, to arrange them according to their behaviour in the presence of different dyes and histological reagents. In doing this, I have avoided pitfalls into which others seemed to have plunged.

§ 1.—*The Neuroglia in Sections Stained by the Method of Bevan Lewis.*

As my earliest acquaintance with the neuroglia was formed from an examination of sections prepared by the fresh method of Dr. Bevan Lewis, I will begin by commenting on the revelations of this process. And I will here take the opportunity of expressing the belief that if others, in particular continental workers, had been more generous in their recognition of this method, past controversies regarding the structure of the neuroglia might have been considerably abbreviated.

So many years have elapsed since Dr. Bevan Lewis gave us his earlier publications, that many others in this country have made themselves familiar with his process and confirmed his observation that, in frozen sections of the fresh brain stained with "blue black" and some other aniline dyes, two kinds of glia cell are displayed, one small, the other large.

The smaller elements vary from 6 to 9 μ in diameter, and have a spheroidal, intensely-stained nucleus, surrounded by an extremely delicate protoplasmic investment apparently devoid of processes. They appear in three situations, irregularly distributed throughout the nerve fibre framework, in regular series around nerve cells, and in more or less regular succession along the course of the blood-vessels.

The larger elements are usually 13 μ in diameter, and are further distinguishable from the smaller by a preponderance of protoplasm over nucleus, by relatively pallid staining of the nucleus, by their flask-like form, and by showing numerous delicate radiating processes, one of which, stouter than the rest and attached to the wall of a neighbouring capillary, is known as the vascular process. In the normal brain such cells occur chiefly in the outermost layer of the cortex and in the white or medullary projection of a convolution. They bear a constant relation to the blood-vessels of the cortex. These are the cells described by Deiters in the year 1865; by many they are called after him "Deiters' cells"; but "scavenger cells," on account of their supposed phagocytic function, and "large spider cells," are other names which have been applied. We shall have frequent occasion to return to these cells, because as a factor in the production of cerebral sclerosis and as a feature in other morbid conditions of the cortex they possess great importance.

§ 2.—*The Neuroglia in Sections Impregnated with Salts of Silver.*

The service rendered to the neuro-histologist by Golgi when he elaborated his well-known process cannot be over-estimated. For the embryologist the method has no equal; with its aid all the beautiful work of Golgi, His, Capobianco, and Fragnito, and others, on the development of the neuroglia has been performed. Similarly, the ordinary histologist will vouch that with this process, or one of the many modifications which have been recommended, a clearer demonstration of, at any rate, the external form of the neuroglial element is obtained than by any other method.

The display in a silvered preparation of the normal cortex is very different from that in a fresh section stained with aniline blue black; in fact, there is some difficulty in correlating the appearances. The elements seen may be briefly described as follows: (1) Cells with short (hence the German name "Kurzstrahler"), thick, mossy, or feathery branching processes roughly coated with the deposit of the silver derivative, one of which is a vascular process. These cells, admirably fitting the designation "spider cell," have the same distribution as the larger elements to be seen in fresh sections, and, although the silver coat alters their general appearance, there is no doubt that the two are identical. (2) Cells with a smaller body, from which numerous delicate, long (hence the German name "Langstrahler"), and clearly-defined processes radiate. A vascular attachment can be found sometimes, but not always. The term "stellate" applies well to the shape of these cells. The white matter is again their elective site, and they are never far removed from a blood-vessel. (3) The third variety of cell was, I think, first described by Retzius, and occurs only in the plexiform layer (Ramón y Cajal) of the cortex, immediately beneath the pia mater. They seem to hang down from vessels in the pia mater, to which they are attached by several processes, including the vascular process; and from the opposite or central end of the body there springs a pencil of branching, delicate, and occasionally moniliform processes.

It is commonly stated that the three varieties of cells whose characters I have just described are all transition forms of one element. This is a statement to which I am inclined to subscribe my belief. In this connection the character they have in common of growing in direct contact with a blood-vessel is most important. For, when we take notice of the point that they all have a different environment, one deposited in the loose substance of the cortex, another in the dense plexus of fibres in the white substance, and the third next the surface of the brain; when, also, we recollect how variable in its chemical reaction to silver salts is protoplasm in general, the statement gains additional justification.

Unfortunately, I cannot leave the silver method without expressing a word of regret. Excellent as the methods of Golgi, Cox, and Ramón y Cajal are as aids to the embryologist and the normal histologist, their fickleness of action condemns them in the eyes of the histopathologist, and I have found them of little value in my examination of sclerosed tissues.

§ 3.—*The Neuroglia in Sections Stained by the Methyl Violet Method of Weigert.*

We appeal next to a method which has received a high measure of praise, because of all methods it alone can pretend to be selective in action. Much faulty reasoning, however, seems attributable to overdriving this selective virtue. Simple constitutional principles appear to have been neglected. It has been forgotten that almost all tissues are chemically and structurally complex, and that to meet this combination there necessarily must be selection within selection. Take the nerve cell as an instance. Step by step we have perfected our knowledge of its structure by discovering that one dye or one means of impregnation selects and displays the cell envelope, another the tigroid bodies, another the neurofibrils, and so on. Accordingly, we find in practice that not one but many methods of staining have to be gone through before we obtain a satisfactory demonstration of the structure and condition of any nerve cells. And surely the same applies to the neuroglia.

I have made this explanation preparatory to saying that I share the opinion of those who refuse to entertain the statement of the late Professor Weigert that the nucleus and the fibrils in the neuroglia cell are chemically different and morphologically separate. The statement is one which has been much discussed, and, although the preponderance of authority is unfavourable, it still finds many adherents. Those, however, who have stained and studied the neuroglia by various methods, and in conditions of health and disease, are forced to conclude that the supposition of the late Frankfort professor rested on a faulty basis. Without denying that the fibrils and the nucleus of the neuroglia cell differ chemically, they will rest their argument on the point that the neuroglia cell, like many other cells, is composed of envelope, nucleus, hyaloplasm, and spongioplasm. Each constituent requires special treatment for its display, and the last is peculiar in that it harbours fibrils, which extend along the processes and have a special affinity for methyl violet. The case, therefore, is one of limited selection. The method affords an exquisite display of the framework of the cell, but fails to show the covering; and I think that this may explain why Weigert held that the neuroglia cell had no vascular attachment, for we have reasons for assuming that this particular process is composed of non-fibrillar protoplasm.

And now a word on a question which has arisen out of the controversy to which I have just alluded. Can the neuroglia fibre live independently of the cell body? Possibly, but that existence is qualified by certain conditions. It appears that the neuroglia cell in the fully-developed form has a definite period of life, that there is a cycle in its history, that in its earliest form it is a nucleated mass of structureless protoplasm, that the formation of its contained fibres is a comparatively late phase, and that in the stage of devolution the nucleus and investing protoplasm disappears, leaving nothing but the fibrils behind. Unless some transition of this nature takes place, it is impossible to explain many forms of sclerosis in which the method of Weigert, and several other methods, display a dense fretwork free from nuclei of any description and composed exclusively of delicate fibrils.

As with the method of Golgi, so with the method of Weigert; as it stands at present it does not commend itself to those investigating the condition of diseased tissues, because, for some unknown reason, it is uncertain in its results, and the risk of spoiling tissue when the supply is limited is a deterrent to its employment; but, as we all know, these imperfections were an admitted source of regret to the illustrious professor at the time of his death.

§ 4.—*The Neuroglia Cell in Sections Stained by the Platinum Method of Ford Robertson.*

Dr. Ford Robertson insists strongly that the neuroglia is not composed of one element, as is generally believed, but of two kinds of cells, of which "the origin, morphology, and behaviour in morbid conditions are entirely distinct." This hypothesis is based on the revelations of a specially-devised method of impregnation with platinum. The elements which Dr. Ford Robertson has discovered, and for which he proposes the name "mesoglia," are blackened by platinum, while the ordinary neuroglia cell is only darkened. They are small cells, with scanty perinuclear protoplasm and three to six delicate processes which tend to branch dichotomously. These processes, compared with those of the ordinary neuroglia cell, are short and sometimes varicose; they form no vascular attachment; indeed, special relation with any other structure has not been determined. Though first observed in the brain of the dog, such cells have been found in man also, and they are said to occur throughout the central nervous system. Some may think that Dr. Ford Robertson's evidence is scarcely strong enough to clinch the supposed mesoblastic origin of these cells; even so, and granted that more information is required, their discovery alone is an important addition to our knowledge of the glia.

I have now exhausted the list of stock methods for the display of glial elements, though in practice other stains and reagents are of contributory service; for instance, I have frequently obtained a beautiful view of the neuroglia as it appears in the white substance of the brain in sections primarily stained for neurofibrils by the method of Bielschowsky; and rubin, as recommended by Kultschitzky, Mallory's hæmatoxylin, and several of the carmine preparations now falling into disuse, may prove convenient in helping us out of difficulties, but none of these stains reveals any elements not demonstrable by the principal methods, and therefore may be dismissed.

§ 5.—*Summary Enumeration of Neuroglial Elements.*

(1) The large neuroglia cells of the fresh method of Dr. Bevan Lewis, corresponding to the spider cells of the method of Golgi, and also revealed by the method of Weigert. Synonyms: large spider cell, scavenger cell (Bevan Lewis), Kurzstrahler (Lenhossek and other German authorities).

(2) The stellate cell of the method of Golgi, also revealed by the method of Weigert, and possibly representing a phase in the development of No. 1. Synonyms: small spider cell, Langstrahler.

(3) The cell next the surface of the cortex, displayed by the method of Golgi, possibly representing a form taken by No. 1 in an unusual environment. Synonym: glia cell of Retzius.

(4) The smaller cell of the fresh method of Dr. Bevan Lewis, sometimes spoken of as a nucleus of the neuroglia.

(5) The mesoglia cell of Dr. Ford Robertson.

CHAPTER 2.—DEVELOPMENT AND GROWTH OF THE NEUROGLIA.

In the year 1885 Golgi told us that he had seen epithelial cells in the chick's spinal cord which he thought gave origin to the neuroglia; these cells lined the neural canal, and from processes which they sent out to the pia mater the neuroglial element seemed to grow.

Since this opening speculation, many others have devoted attention to the subject, and, extending Golgi's observations, they have obtained evidence which suggests that other neuroglial elements may spring from the primitive epithelial cells of the central canal as well as from cells of the neural canal, and others, again, from mesodermic elements, penetrating the nervous system along with the embryonic blood-vessels. In short, our information leads us to suppose that the neuroglia is descended from both epiblast and mesoblast.

And now, referring the reader desirous of further details on the early development of the neuroglia to the monographs of Golgi, His, Capobianco and Fragnito, and Ford Robertson, we can pass to a matter of more immediate interest, the history of the neuroglia in the later periods of life.

In this connection we seek information more precise and comprehensive than that already supplied on the series of tissue changes in the developed brain which end in sclerosis. And when we analyse our requirements we find that they nearly all revolve round one question, whether this sclerosis is brought about by a proliferation and hyperplasia of pre-existing elements or by the formation of new elements? This question I shall try and answer.

Thinking that profit might accrue from closely watching the sequence of events in the tissues surrounding cerebral lesions, both those arising in nature and those of artificial production, I carried out a special series of observations. For the artificial lesion I took a number of white rats, plunged a red-hot needle into the brain of each, and then killed the animals and examined the lesion after periods varying between one and twenty-one days. The brains of a series of cases of cerebral softening in the human subject, which I have collected and placed in the museum at Rainhill Asylum, afforded rich material for examining the changes in a natural lesion; more than this, the latter study safeguarded me against the objection that my results might have been invalidated by structural differences between the human brain and the brain of the rat.

In narrating the conclusions drawn from this examination, it will be convenient to take up in turn the varieties of cells met with and trace their life history. This will embrace an account of cells which I will classify as follows:—

(1) Large mononuclear phagocytes.

(2) Polymorphonuclear leucocytes and compound granular cells.

(3) Eosinophile cells.

(4) Lymphocytes or lymphoid cells.

§ 1.—*The Large Mononuclear Phagocytes.*

By the large mononucleated phagocytes (the designation is borrowed from Beattie) I mean cells which are, without doubt, the most important constituents in the conditions with which I am dealing. They are considerably larger than a leucocyte; their nucleus is large, crescentric, or reniform, and usually eccentric; it often touches the margin of the cell, and is supplied with chromatin, scanty and sometimes knobbed beneath the nuclear membrane. A nucleolus is almost always recognisable; the body protoplasm is scanty, with most dyes it remains homogeneous, but with Unna's alkaline methylene blue it stains darkly and shows some fine granules. These are the characters in the first stage of development, and I think it will not be denied that such cells are identical with cells described by many other observers in different inflammatory conditions, and called variously *large mononucleated leucocytes* (Metchnikoff), *infiltration cells* (von Marschalko), *pseudo-plasma cells* (Hodara), &c. Let us follow the history of these cells. In the early stage of a lesion, be it a hæmorrhage, a softening, or an artificial destruction, they are not at all prominent; if, however, we search along the vessel walls, particularly along the walls of vessels lying just without the focus of destruction, we may find what I take to be the originals of these cells. In the first state they appear to consist of nucleus alone; at any rate, the investing ring of protoplasm is extremely delicate, and it is at this stage that we get a hint as to their origin, for they are seen in such intimate connection with the walls of capillaries that it seems certain they are derived either from the endothelium lining these vessels or from contiguous lymphatics. It is not clear how they multiply, but, although I have not seen mitosis, I believe they divide by this method. Detached from the vessel wall, I imagine they next migrate to surrounding tissues, and their further development belongs to another stage.

In a similar lesion about six days old the conditions are changed; difficult to find previously, the cells in question now seem abundant, but this apparent increase in number is due in no small measure to an increase in size; they approach the mature state. The nucleus remains as it was, but is so eccentric that it often looks as if it were on the point of breaking away from the cell. The scanty ring of body protoplasm is supplanted by a copious, pale-stained, homogeneous, or finely-stained mass, from the serrated edges of which many coarse processes extend, and it is significant that one of these processes establishes a direct connection with the nearest capillary or arteriole. Next, after a few days, the cell becomes reduced in size and the protoplasm of the processes condensed and converted into deeply-stained fibrils.[1]

Cells of this character may be recognised for weeks and even months after the preliminary disturbance, and may be studied conveniently in the tissues surrounding a patch of "yellow softening," where they are present in great abundance. In such material I have observed changes in these cells which have an important bearing on the histogenesis of sclerosis. Thus, as the cells grow older they lose their distended globose figure, and their processes become larger and more attenuated. And in this stage one is struck by the resemblance they bear to the larger neuroglia cells of the normal brain; indeed, the appearances afford proof, almost positive, that the commoner forms of sclerosis are brought about by the deposition and metamorphosis of new elements, and not by the proliferation of pre-existent cells; also they strengthen our belief in the statement of Fragnito and Capobianco, that in the course of development the ingrowing blood-vessel is an important channel for the convection and introduction of the nuclei from which the neuroglia grows.

Passing on to the final stage in the history of these cells, displayed, for example, in the walls of an excavation resulting from old softening, the case is one of still further shrinkage. As an outcome of contraction and failure of nutrition from autochthonous vascular obliteration, the body and nucleus of the cell either disappear or become changed into small, round, deeply-stained nuclei; the processes gain independence, but remain as attenuated and filamentous fibrils contributing to the formation of a network of great density. Touching the nuclear remnants, it is possible that they represent what are often called the nuclei of the neuroglia, that is, deeply-stained, circular, or oval nuclei; and, as it is absurd to think of a nucleus without function, it is possible that these bodies persist to exert some influence in the maintenance of adjacent neuroglia fibres.

---

[1] When making these observations I had forgotten for the time being that Dr. Watson had made a study, wanting nothing in exactitude, of the developing neuroglia in the brain of the juvenile general paralytic. It is satisfactory to find, however, that what is written above is quite in harmony with Dr. Watson's fuller and more precise account. (*Archives of Neurol. London County Asylums*, ii., 1903.)

Concerning the function of the cells, the life history of which has just been given, I have no hesitation in agreeing with Beattie and others in saying that they possess phagocytic properties, because in the swollen state it is common to see particles of blood pigment, the products of myelinic disintegration, and even nucleated bodies (leucocytes or lymphocytes) included in the cell body. I do not think, however, that they play such an important *rôle* in the absorption and removal of diseased and foreign matter as do the leucocytes; the part they fill in a later phase, that of binding and strengthening the zone of substance bordering the destroyed area, is of greater importance. Then, speaking of the function of neuroglia cells in general, it is in accordance with these data to suppose that in certain phases a cell may act, as Bevan Lewis suggests, as a scavenger, while in others it may serve as connective tissue, and in the latter capacity coincidently it might fulfil the part of an isolator (Eurich, &c.). That these cells are directly connected with the lymphatic system is for me difficult to believe, but that in an indirect manner they serve as drains for lymphatic fluid is readily credible, and in this capacity it seems of little moment that the vascular process should be solid instead of canaliculated, a point out of which some have made capital in criticising this hypothesis. Here the siphonic action of a piece of string or worsted is a physical truth to be held in view.

Satisfied that the cell described is the most important element in the process of repair as it affects the brain substance, only a short account of other cells met with is called for.

§ 2.—*The Polymorphonuclear Leucocytes and Compound Granular Cells.*

In all artificially-produced and many natural lesions, in early and in late stages of the process, these cells, lying collected within and in the neighbourhood of obstructed blood-vessels, are prominent objects. So often are they choked with masses of blood pigment, altered proteids and detritus, that there can be no doubt they are more active agents in the removal of products of degeneration and disease than the large mononucleated cells; moreover, in this case they re-exhibit the phagocytic properties they are known to show in those conditions in which micro-organisms are the principal objects for removal.

The constant presence of these polymorphonuclear leucocytes in the cases of cerebral lesion in hand has naturally excited the suspicion that one might be a transition form of the other; more than this the frequent occurrence of collections of kindred leucocytes (compound granular cells, as they are sometimes called) along the blood-vessels in patches of disseminated sclerosis and slowly-progressing myelitis, appears to favour the possibility that these are the originals of glial or connective tissue cells. I have therefore taken considerable pains to arrive at the truth on these points, and I can say now that, while my specimens provide abundant illustrations of swelling, fragmentation, and dissolution of these elements, all proof of their persistence and metamorphosis into the large mononucleated astrocyte is absent.

§ 3.—*Eosinophile Cells.*

The coarsely-granular eosinophile cells of the blood seem to play no part of importance in the process of repair.

§ 4.—*Lymphocytes or Lymphoid Cells.*

The plasma cells of von Marschalko have been attracting attention lately, because it has been stated that they are pathognomonic of general paralysis of the insane, and, as they are present in some of the conditions of which I shall treat, they must be noticed. According to von Marschalko, they are derived from the lymphocytes or, as he calls them, the lymphoid cells of the blood, and they are described as having scanty, non-granular protoplasm, and an eccentric oval nucleus showing several chromatin granules and a nucleolus.

These cells resemble in many particulars the large mononuclear phagocytes in their earliest state of development, and whether they are derived from the endothelium of blood-vessels or from lymphocytes, it is my belief that they are identical with these phagocytes.

CHAPTER 3.—TUBEROSE SCLEROSIS.

Synonyms.—*Sclérose tubéreuse ou hypertrophique du cerveau (Bourneville). Multiple tuberöse Sklerose des Gehirns. Hypertrophic nodular gliosis (Sailer). Neurogliosis gangliocellularis diffusa (Hartdegen and Neurath). Anoia (Sherlock).*

First described by Bourneville in the year 1880, tuberose sclerosis is one of the most extraordinary diseases with which the neuro-pathologist has to deal. Not only are the potato-like (hence the name tuberose) masses of sclerosis in the brain unique in kind, but the accompanying affections of parts related neither to the brain nor to one another are as singular as they are difficult to explain.

Sailer found twenty-eight cases reported in medical literature. I have not been so fortunate; still, I have read accounts of twenty; and on running over the salient clinical features, the first point which struck me, one which I desire specially to emphasise, is that no less than seventeen suffered from idiocy or imbecility, and that, even in the remaining three, congenital mental weakness could not be excluded. This complication, remarkable in itself, suggests an intrauterine origin for the cerebral disease which underlies it.

Convulsions, identical with those of ordinary ideopathic epilepsy, form a second clinical manifestation, and although unmentioned in some reports, seem to have been invariably present in all the best recorded cases.

The frequent and, I believe, constant occurrence of the peculiar and rare skin affection, adenoma sebaceum,[1] is a third sign, which, taken in conjunction with those just mentioned, as well as the youth of the patient, may lead to a diagnosis of the disease during life. The age limit for the occurrence of this disease is 25 years.

Great patches of indurated cortex, projecting nodules in the ventricles of the brain, and fibroid subcapsular tumours in the kidney, complete the extraordinary combination.

While most writers make no attempt to explain the remarkable complex of diseases, the pathology of the cerebral changes has been the subject of much speculation. Taking a retrospect we find that the view meeting most support—it is the only one which brings all the somatic changes into line—is that we have to deal with some obscure developmental anomaly of the nervous system. And touching the time of life at which this arises, it is probable that the process is in activity at birth, because Hartdegen has recorded a case in a newly-born infant. It appears also that although the germs may be sown earlier, they cannot be active before about the seventh month of intrauterine life, the month when the plan of the sulci and gyri is laid down. We are entitled to this assumption because we have learned from our studies of porencephaly that when the destructive lesion peculiar to that disease occurs prior to the seventh month, the effort on the part of the growing cerebrum to make good the deficiency results in distortion and a characteristic fan-shaped and bizarre arrangement of sulci and gyri around the lesion. When, on the other hand, the lesion occurs after the seventh month the gyri and sulci surrounding the destroyed part can all be identified and the arrangement is orderly. Now, in tuberose sclerosis, in spite of the deposit of great masses of fibroid material all over the brain, there is no disturbance of external morphology. Therefore, knowing the distorting effects of cicatrisation, we cannot suppose that the sclerotic process assumes activity before the time I have mentioned.

---

[1] Adenoma sebaceum is a congenital affection having a predilection for the skin covering the bridge of the nose and the naso-labial folds; it is said to consist of hypertrophy of the sebaceous glands, which appear as minute, shotty nodules, lifting the epidermis and projecting above the general surface level. Pringle gives a good and succinct account of the disease in "Quain's Dictionary of Medicine" (3rd edition, London, 1902), and mentions the interesting facts that the observed cases have generally been afflicted with mental disease, and that other congenital diseases of the skin, such as warts, moles, nævi, and fibromata, have commonly co-existed.

Other views on the genesis of the condition can be soon dismissed. Fürstner and Stühlinger, in assuming that a leptomeningitis arising in intraüterine life or early youth is the cause of a chronic gliosis, not only take an unwarranted liberty, but fail to account for the other somatic changes.

Koch's explanation seems to rest solely on a coincidence. In his case, thickening and adhesion of the pia mater beneath an old fracture of the parietal bone betrayed a difficult labour, and the application of forceps, which he was led to regard as a cause of the sclerosis; but we have had so many cases of tuberous sclerosis not so complicated that this suggestion cannot be entertained.

The fourth view, that of Scarpatetti, puts the onus of the disease on syphilis in the parents, "as a result of which multiple hæmorrhages affect the brain of the offspring at an important period of intrauterine development and call forth an extremely chronic form of reactive inflammation." This, again, fails to explain the invariable coexistence of the changes in the skin and kidneys; moreover, the constancy of the syphilitic taint has yet to be proved.

A microscopic examination seems to have been carried out in most of the recorded cases, and all observers agree in stating that the brain masses are composed of a dense network of proliferated neuroglia, choking and destroying the nerve-cell and nerve-fibre elements; only three writers, Sailer, Hartdegen and Neurath, mention the presence of scattered but exquisitely formed giant or ganglionic nerve cells. Such cells are most interesting constituents of the masses; they occur in my specimen, and, since in a preliminary examination I overlooked them, I am inclined to think that others may have done the same. More than this, I think that they are constantly present. Accordingly, Hartdegen and Neurath are misleading when they call the disease "glioma gangliocellulare," for, although this name correctly conveys the histology of the condition, when unqualified it creates the false impression that it is a disease distinct from tuberose sclerosis. Besides, we must not forget that there is another altogether different form of cerebral sclerosis associated with the presence of ganglionic nerve cells.

I will now proceed with the account of my case.

Clinical History.

The patient, a male, was admitted to Rainhill Asylum at the age of 6 years, and died therein at the age of 16. He came from a workhouse, and, unfortunately, no information regarding his family history could be obtained. Examination, on admission, revealed no sensory, motor or nutritional abnormality, but adenoma sebaceum was noticed affecting the skin covering the bridge of the nose and the naso-labial folds. Though congenital syphilis could not be excluded, the teeth were in good condition, and there were no outward indications of this disease. His mental state was one of idiocy. When spoken to he lifted his head, but his expression proved that he did not comprehend language; nor could he use it. He would pick up objects and handle them in an aimless way, but did not resent their removal, apparently forgetting that he had just had them in his possession. He was ill-tempered, restless, given to wandering about in an objectless manner, and to distorting his body and making grimaces.

He suffered from epilepsy, and this is the sequence of events observed in a fit occurring soon after admission. The opening cry was succeeded by conjugate deviation of the eyes to the right, and by dextriversion of the hand. The spasm was more marked in, and almost confined to, the muscles of the left side. When recovering, he placed his hands to his mouth and moved his jaws as if chewing at a bone. There was no expulsion of fæces or urine.

Beyond learning to understand and carry out simple instructions, the boy made no mental progress; physically, on the other hand, he developed normally, with this exception, his cranium failed to expand in proportion to his body growth, and left him distinctly microcephalic. He continued to suffer from epilepsy, averaging five fits a month, a number which dropped to one a month during the last year of life. Five months before death he was treated in the infirmary for an obscure attack of pyrexia. This was transitory, and, at the time, regarded as trivial, but when he returned to the care of his usual attendant it was noticed not only that he was more apathetic and irritable than before, but that he was quite blind. (The necropsy proved that this attack was due to occlusion of both posterior cerebral arteries.) After this he gradually failed, and death was ascribed to exhaustion from epilepsy.

It is noteworthy that the record of the case contains no reference whatever to tremor, to paresis, or to any similar manifestation which we might expect to be associated with the cerebral sclerosis.

Anatomical Examination.

The Brain.—The brain was below the average in size and weight, the encephalon scaled 1,210 grammes, the right hemisphere 476 grammes, the left hemisphere 500 grammes, both unstripped, and the cerebellum with the pons and medulla 157 grammes. The occipital lobes were reduced in size by bilateral adventitious softening, to be described presently; but there was no lobar deformity or disparity which we could set down to defective development, and, in accordance with my introductory remarks, the disposition of gyri and sulci was free from striking peculiarity. (Fig. 1.)

The sclerosed masses characterising the disease did not obtrude, indeed, at first glance the surface appeared healthy; on closer inspection, however, numerous patches were observed, twelve or more in each hemisphere, some circular, others irregular in form, ranging between 1·5 and 5 cm. in diameter, and universally distributed, although chiefly occupying the frontal lobes of both sides, and the parieto-temporal region of the right side. These patches were characterised by an extraordinary firmness—they were little softer than costal cartilage—by a paler tint and a more granular aspect than surrounding parts, by projecting slightly above the surface level and by being coated with pia, not adherent, but poorer in vessels and more delicate than the rest. It was noticed, further, that some of these patches presented slight central umbilication, that the proximal wall or lip of bordering sulci was bulged so as to operculate the

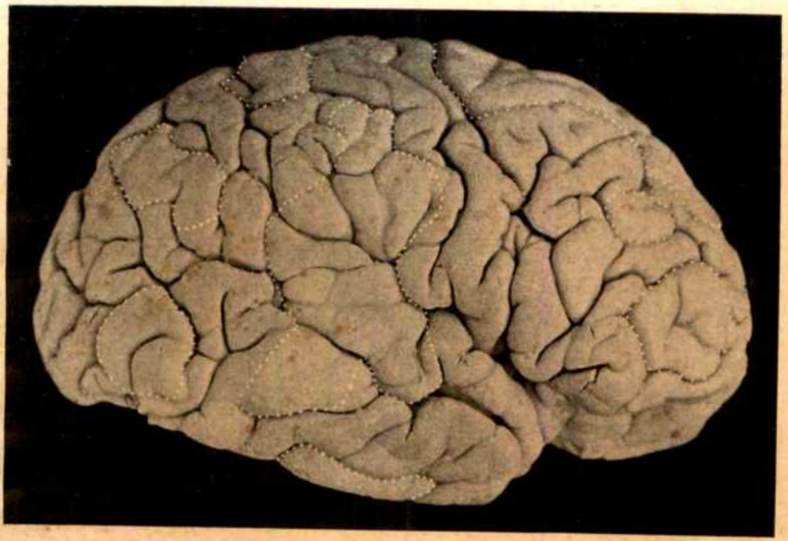


FIG. 1.

Photograph of the outer surface of the right cerebral hemisphere in the author's case of tuberose sclerosis. The distribution of the sclerosis is indicated by the dotted lines. The patches cause no disturbance in the plan of the convolutions.

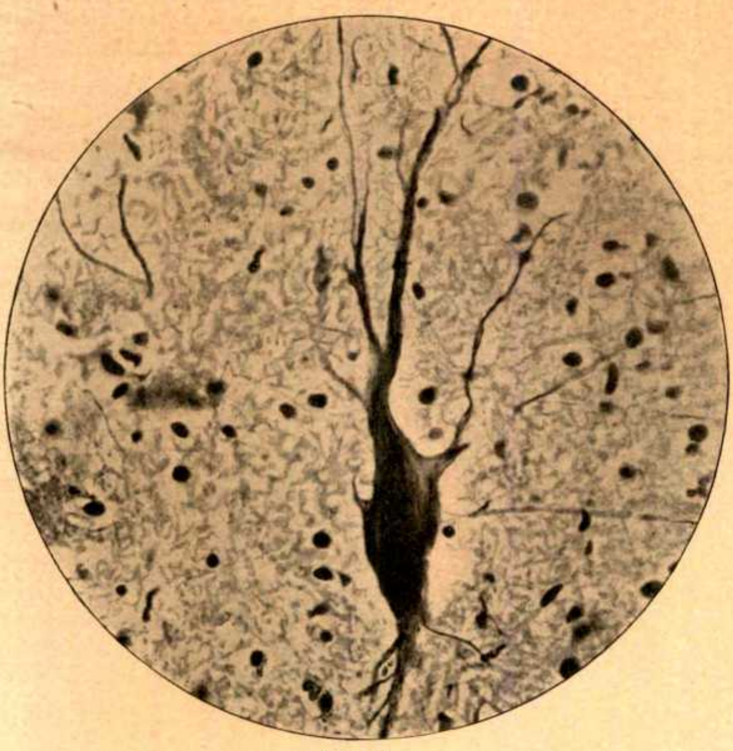


FIG. 2.

Microphotograph of a giant nerve-cell from one of the sclerosed patches. × 400. In the original specimen, stained by Bielschowsky's method neuro-fibrils can be plainly seen.

opposite distal wall (a manifestation of cortical hypertrophy), and that in most cases the outline was sharp, though occasionally the passage from cartilaginous firmness to normal consistence was gradual. On section, the cortex was manifestly increased in depth, and the depth of invasion of the white substance was not in proportion to the surface extent; indeed, I think it may be correctly said that the sclerosis was mainly confined to the cortex and that only the immediately subjacent white substance was affected. This applied, at any rate, to most of the deposits; therefore, in regard to morphology, while the term "tuberose" aptly expressed the outward appearance of the masses it did not fit the sectional view.

So much for the cortical deposits. I have now to describe another variety of neoplasm seen on opening the lateral ventricles, which were dilated. These growths were of quite a different character, small (about the size of a split pea), white and very hard, they projected above the surface and were distributed along the lines of the venules, coursing over the basal nuclei and draining into the main vein of the corpus striatum. Others do not seem to have noticed this relation to the venules, but it is important and will be referred to again in discussing the histogenesis of the disease. One deposit, larger than the rest and situated beneath the ependyma lining the anterior horn of the lateral ventricle, appeared at first sight to be of different nature, but microscopic examination proved that this was not so. Subependymal growths of this description have been repeatedly observed in "tuberose sclerosis," and their occurrence quite away from the surface of the brain suggests that the original germs of the disease are sown elsewhere in addition to the cortex.

The occipital shrinkage previously alluded to was the result of bilateral occlusion by thrombosis of the posterior cerebral artery; the subsequent softening comprised the "area striata" (the area presenting the line of Gennari) and a portion of the subjacent lingual lobule. The affected parts were yellowish-brown and much contracted. Seeking a cause for the thrombosis, we found sclerosed masses attacking the cortex in the stem of each calcarine fissure; they bulged into the fissures, and their pressure must have directly induced stasis and coagulation in the arteries. Such a bilateral softening is rare, and explained the patient's sudden blindness.

The cerebellum, pons, medulla, cranial nerves, spinal cord, and all remaining structures pertaining to the nervous system, appeared normal.

In the trunk the condition of the kidneys attracted attention. In each quite a dozen firm, round, white growths, 1 to 5 mm. in diameter, were seen projecting above the surface level; they were implanted in the cortex, but not deeply, as some came away with the capsule when it was stripped; they did not occur in the medullary substance, and otherwise the kidneys were healthy. Similar growths have been constantly found in the kidneys in other cases.[1]

In the left lung we found two wedge-shaped patches, solid and uniformly gray in colour, which we took for resolving infarcts, but there was no zone of congestion around and no heart disease[2] to account for them, so that our diagnosis may have been at fault.

The remaining organs were free from special disease.

Microscopic Examination.

(1) *Sclerosed Cortex.*

(*a*) *Nerve fibres.*—Sections which I have treated by the method of Wolters-Kulschitzky show profound want of fibres. There is no trace of a zonal layer, delicate fibrils are scattered about in the supraradiary layer, a line of Baillarger is barely discernible, the interradiary spaces are swept almost bare, only a few fibres remain to mark the position of the radiations of Meynert, and even these seem to be bereft of their myelinic investment. Such changes are more pronounced in the centre than at the periphery of the masses.

---

[1] In Sailer's case a large "adeno-sarcoma" of the kidney was found, but I am inclined to doubt the correctness of the microscopic diagnosis.

[2] Ugolotti found a "fibro-myoma" in the heart muscle.

(*b*) *Nerve cells.*—It might be supposed that nerve cells could not exist in the presence of such profound tissue metamorphosis; they do, however, and their nature and condition demands careful consideration. There is, of course, a fall in numerical representation, and it varies in relation to the march of the sclerosis, being greatest in the most severely sclerosed parts, but nowhere is the cell destruction complete. It appears that the first cells to go are those of small size, namely, cells in the first, second, and fourth (stellate) layers. Some larger pyramids and some fusiform cells remain to the end, but while they retain their erect position and their apical and basal processes, their bodies become attenuated and their reaction to stains changes. With Nissl's method the protoplasm stains deeply and homogeneously, and Bielschowsky's process displays no neurofibrils.

Mention of neurofibrils leads on to another most extraordinary feature of this disease, one to which I have already briefly alluded; it is that the sclerosis is associated with the deposit or evolution of aberrant nerve cells, cells which in point of size and shape are akin to the large multipolar "motor" cells of the normal precentral cortex, and for which, therefore, the designation "giant" or "ganglion" cell is appropriate. (Fig. 2.) Such cells are not numerous, only one or two may be discovered in a section measuring two square centimetres; they affect no particular cortical level, at times being found near the surface, at others in the depths, and occasionally even in the subjacent white substance; they may occur in groups, but it is commoner to find them solitary. Structurally they are peculiar, inasmuch as the method of Nissl displays a protoplasm devoid of all chromophilic elements, non-pigmented, and practically homogeneous, while the method of Bielschowsky proves the persistence of neurofibrils.

It is a point of interest and importance that cells of this description have been recognised in all the masses examined, and, whether growing in the frontal, central, parietal, temporal, or occipital region, they have presented a uniform appearance.

(*c*) *Matrix.*—Examining a piece of tissue from the centre of one of these indurated areas, we find a matrix composed of a dense network of indefinite structure, a tissue showing neither nuclei nor distinct fibres, and one which is probably the outcome of a past neuroglial proliferation. Active neuroglia cells (methods of Weigert and Bielschowsky and staining with rubin) vary in abundance in different parts of the deposit and in different deposits; few are found in the central portions of the masses, nor are bodies which we can call nuclei of the neuroglia present in this situation. At the lateral edge of the masses, however, particularly around the gland-like structures to be described presently, and all along the surface of the cortex, I have found cells of glial nature in great abundance. Some of these are shown in the accompanying drawings (fig. 3), and attention is invited to their peculiar character, especially their great size, their succulent bodies, and their coarse processes.

(*d*) *Blood-vessels.*—Nowhere are the blood-vessels numerous, and those seen have normal, thin walls.

(*e*) *Other structures.*—I have now to describe some structures which have an important bearing on the histogenesis of the masses, structures so peculiar in themselves, and so foreign to the cortex cerebri, indeed, to any part of the nervous system, that I consider it necessary to illustrate one (fig. 3). They are found at the peripheral or growing edge of the deposit, and may be described as tubular glands, leading from the surface inwards for a distance of about 1 mm., and ending in blind alveolar expansions. Not having studied them in serial sections, I cannot tell definitely whether they branch, but I think they do. The lining cells are of columnar form, pallid, and finely granular, and they contain a well-stained nucleus. The basal membrane is indistinct, but "centro-acinar" cells (Langerhans) are present in the lumen of the tube from end to end. These "glands"—so I suppose we must call them—are not numerous, for although I saw one or more ducts cut transversely in almost every section, not often did I find the mouth and whole length of a gland as here represented. But while "glands," such as I have described, are only found

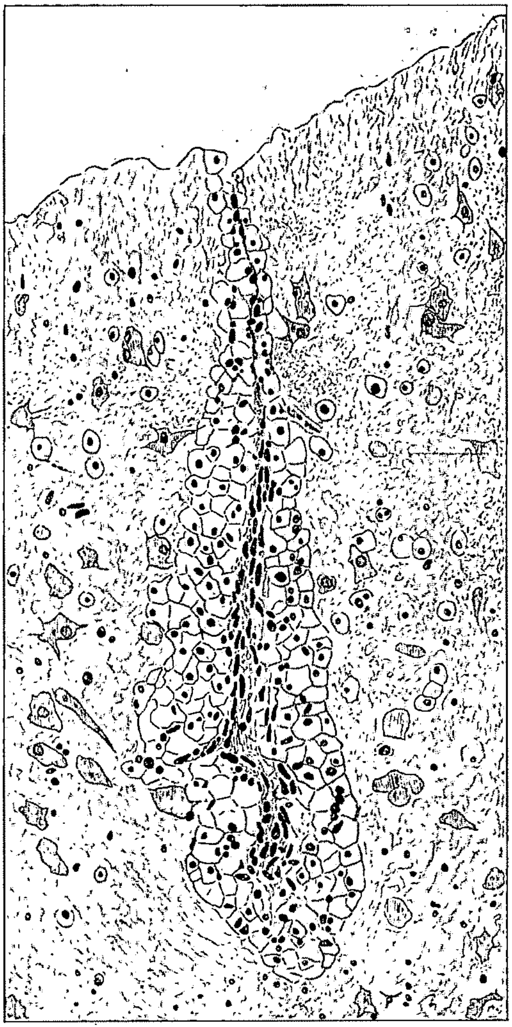


FIG. 3.

Tuberose Sclerosis.

A camera lucida drawing of one of the remarkable gland-like structures described in the text; it leads down from the surface, is tubular, ends in a blind alveolar expansion, and is lined with columnar cells.

Young neuroglia cells are to be observed in the neighbourhood. (Shaded in the figure.)

Beilschowsky's stain. Magnification × 200.

in what I take to be the growing part of the deposits, at the periphery, indications are not wanting that the remainder has been permeated at some time by similar structures, for dotted about in all situations are circular or oval cells 10 to 15 μ in diameter, without processes, finely granular, and with a deeply-stained nucleus 2 to 3 μ in diameter. These I take to be glandular vestigia, not only because they resemble gland epithelium cells more than anything else, but because their number and arrangement varies in different parts of each deposit; in parts where the cortex is of cartilaginous hardness, and the process apparently most advanced, such cells, though present, are not numerous; in other growths which are softer, and apparently of more recent origin, they occur in abundance; moreover, reminiscent of the tabular arrangement, several cells are occasionally seen in line and in apposition.

(2) *The Subependymal Growths.*

The subependymal tumours have not the same microscopic characters as those in the cortex. A single layer of ependymal epithelial cells coats the surface. Immediately below, separating the surface from the main growth, is a shallow layer, composed of densely interwoven delicate fibrils, and containing short lines and groups of ependymal cells. In this layer runs the vein which the growths follow; it looks distended, but the wall is healthy, and so likewise are the accompanying arteries.

The basis of the growth is composed of a more or less open network of coarse fibrous tissue (much too coarse to be called glial tissue), and a remarkable appearance is imparted by an abundance of corpora arenacea, like those seen in a choroid plexus which has undergone calcareous degeneration. Small vessels are numerous, and, since they nearly all have thick calcereous walls, and since where they are most numerous, so also are the corpora arenacea, I think there can be no doubt, as has been inferred by previous observers in other diseases, that there is a developmental relation between the two.

Of nerve cells there are none; indeed, in all the small ependymal growths nucleated cells of any description are scarce. The larger ependymal growth, however, which I have especially referred to, contains, and in fact is almost entirely composed, of cells so remarkable that I figure them (fig. 4). They are elongated and fusiform, and have homogeneous protoplasm and one to three oval nuclei. It is difficult to arrive at a conclusion concerning the nature of these cells, but I hazard the opinion that they are of endothelial origin, and this on account of their resemblance to cells seen in growths regarded as endotheliomata. The difference in appearance between this and the smaller ependymal growths does not necessarily signify a different origin. I take it that the smaller growths are the older, and that they have undergone fibrous and calcareous degenerative changes.

(3) *The Tumours in the Kidneys.*

On microscopic examination these tumours are found to be sharply circumscribed, but not encapsulated. They retain no renal structure, and are composed for the most part of long nucleated cells, smaller, but otherwise not unlike those seen in the larger subependymal cerebral growth. Bloodvessels are numerous, and many show thickening of the adventitia.

Reflecting on the remarkable complex of changes, naked eye and microscopic, furnished by this disease, interest at once turns on the question of histogenesis. And, in considering the origin of the various growths, weight attaches to the determination of the nature and source of several products revealed by the microscope; first, the acinoid structures, which I seem to have been the first to recognise, in the masses of sclerosis on the surface of the brain; secondly, the giant nerve cells finding a place in the same masses; thirdly, the cells composing the intraventricular and the renal growths; and, lastly, the components of the cutaneous deposits.

In taking a retrospect of the propositions which have been advanced to account for the cerebral growths, I

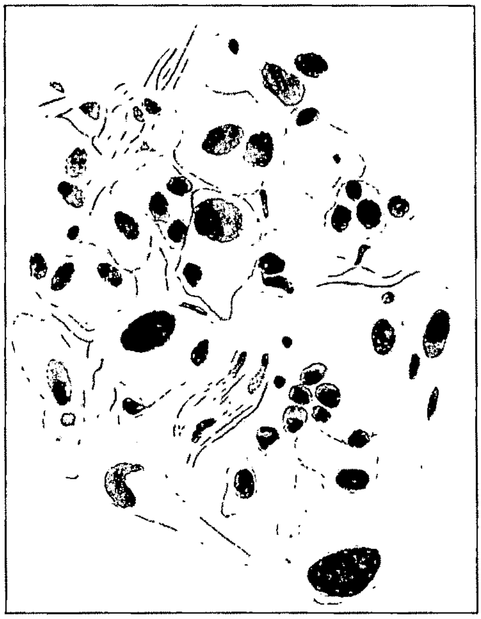


FIG. 4.

Tuberose Sclerosis.

A drawing of some of the endothelioid cells in one of the subependymal ventricular growths.

Silver method of Bielschowsky. × 500.

indicated the certainty that the morbid process, whatever it be, must be active during intrauterine life, although not before the seventh month. It is likely, therefore, that we have to deal with some aberration or disturbance of development, and along this line we will pursue our inquiry.

The preliminary point as to which of the three embryonic tissues, epiblast, mesoblast, or hypoblast, is selected for affection we can soon dismiss, not because the settlement of the point is unimportant, but because the tissue giving rise to one of the organs in which we are interested, the kidney, is undetermined. We know that the nervous system and the skin spring from epiblast, but which layer provides the kidney is still unsettled. Hence we cannot group the affected organs and parts into one category, stating that they are of common embryonic origin. Nor is it necessary, for, in describing the histology of the renal and the intraventricular growths, I insisted that the principal cell constitutents of both had characters reminiscent of cells occurring in endothelial neoplasms; likewise in the case of the growths lodged in the cerebral cortex, I pointed out that gland-like endothelial expansions were present, and were probably the cause of the neuroglial proliferation. This structural correlation is of great importance, in fact, it affords a clue to the solution of our problem, because if my primary assumption that all these growths have an endothelial basis is correct, it only remains to find the source of the endothelial elements. We are not without a guide. I think that the lining cells of blood vessels—probably veins—or of perivascular lymphatics will supply our wants, and such a source, in explaining the occurrence of kindred growths in various organs and parts, related to one another neither spacially nor developmentally, will overcome one serious difficulty.

While advancing my arguments for the vascular origin of these endothelial elements, it will be convenient to mention a few points which annul a possible alternative proposition, that they arise from endothelium-clad membranes. In the first place, a relation between the growths in the cortex cerebri and the arachnoid endothelium might be suggested. Of this I will say merely, there is no evidence: there is no thickening, no adhesion, nothing to favour such a relation. On the other hand, the growths in question arise in a part which is particularly rich in arterioles, venules and perivascular lymph spaces; moreover, when the specific constituents to which I have drawn attention, the gland-like structures, are seen on longitudinal section, the arrangement of cells lining the mouth and the neck is singularly reminiscent of what one has often seen in cerebral perivascular channels.

Secondly, the intraventricular growths might be referred to a proliferation of cells of the ependymal lining, but, as I have shown, there is no indication of ependymal hyperplasia, and, on the other hand, there is clear and suggestive evidence of an intimate relation with subependymal venules.

Thirdly, the peritoneum might be thought to give rise to the renal growths, but this is in opposition to the observation that growths occur on the dorsal surface of the kidney, well away from the peritoneum, while, in support of my contention, the close relation of these growths with the venæ stellatæ cannot be gainsaid.

Lastly, in the case of the cutaneous tumours the endothelium of sebaceous glands is to be considered. At the same time, it must be remembered that these growths affect the most vascular part of the face, also, that in some histological reports written by dermatologists, the "nævoid" character of the growths is emphasised.

Therefore I feel justified in asserting that while the relation of the various growths to some endothelium-bearing structure is suggestive, that relation is neither so intimate nor so convincing as it is to the blood-vessels.

If, then, it be allowed that the endothelial cells forming the basis of these deposits is derived from blood or lymph channels, what is the process of evolution, and why should it be confined with such remarkable regularity to the same organs and parts? These are questions which for the present must remain unanswered. We may surmise that the activity of the germinal elements giving rise to the growths has been deferred by some error in development, and that they have continued to grow in an abortive and spurious fashion long after their appointed term of expansion and metamorphosis; and we may imagine that development has been disturbed by some unknown intrauterine, infective disorder, having, like scarlet fever and many other diseases and conditions which could be named, a predilection for unrelated organs and parts.

But apart from these speculations, I submit that the data I have set down favour my primary assumption that the complex of changes characterising this disease is the outcome of some evolutionary aberration or disturbance arising during the last few months of fœtal life, affecting the endothelium of blood-vessels or lymphatics, and resulting in a form of structural hyperplasia and heterotopism.

I have added the word heterotopism to this definition to cover the occurrence of giant nerve cells in the growths affecting the cerebral cortex, and my concluding remarks will be devoted to a consideration of these elements. To say the least, their presence is puzzling, and unfortunately our knowledge of the development of the nerve cell is not advanced sufficiently to admit of a definite explanation regarding their origin. I have noticed, however, in my studies of the neuroglia cell in the growing brain, that at a certain stage of development—prior to the formation of the processes—it is difficult to distinguish between the young neuroglia cell and the young nerve cell; and, although there is no developmental evidence to show that under normal conditions nerve cells, like neuroglia cells, are derived from elements pertaining to the vascular system, still the similarity in structure during development is suggestive, and I cannot help thinking that an embryonic disturbance might lead to an occasional exchange of form, to the aberration in development and the heterotopism of which this disease provides examples. Finally, although these nerve cells are most interesting constituents of the cortical masses, there is not the slightest evidence that they are active factors in the production of the gliosis; on the contrary, their presence favours the original assumption that tuberose sclerosis is the ultimate manifestation of some evolutionary aberration or disturbance.

Tuberose Sclerosis.

Berdez. "De la sclérose tubéreuse du cerveau." *Ziegler's Beitr. zur path. Anat.*, Band 17.

Bourneville. "Sclérose tubéreuse des circonvolutions cérébrales, idiotie et épilepsie hemiplégique." *Arch. de Neurologie*, 1880.

Bourneville. "Sclérose cérébrale hypertrophique ou tubéreuse, compliquée de meningite." *Progrès Médicale*, 1896, p. 129.

Bourneville. "Idiotie et épilepsie symptomatiques de sclérose tubéreuse ou hypertrophique." *Arch. de Neurologie*, 1900, July.

Bourneville et Bonnaire. "Sclérose tubéreuse ou hypertrophique des circonvolutions cérébrales. *Bull. de la Soc. Anat.*, 1881, p. 180.

Brückner. "Ueber multiple tuberöse Sklerose der Hirnrinde." *Archiv. f. Psychiatrie*, 1882.

Förstner und Stühlinger. "Ueber Gliose und Höhlenbildung in der Hirnrinde. *Archiv. f. Psych.*, Band 17, p. 1.

Hartdegen. "Ein Fall von multipler Verhärtung des Grosshirns nebst histologisch eigenartigen harten Geschwülsten der Seitenventrikel bei einem Neugeborenen." *Arch. f. Psychiat.*, Band 11, p. 117.

Koch. "Fall von Idiotie." *Neurol. Centralb.*, 1887, No. 3.

Neurath. "Beitrag zur postinfectiöse Hemiplegie in Kindesalter und zur pathologischen Anatomie des kindlichen Centralnervensystems (Neurogliosis gangliocellularis diffusa)." *Arbeiten aus Prof. Obersteiner's Labor.*, 1899, Heft 4.

Pollati. "Congenitale multiple Herdsklerose des Centralnervensystems; partieller Balkenmangel." *Archiv. f. Psychiat.*, 1882, p. 157.

Pozzi. *L'Encephale*, 1883, No. 1.

Sailer. "Hypertrophic nodular gliosis. *Journal of Nervous and Mental Diseases*, 1898, vol. xxv., p. 402.

v. Scarpatetti. "Zwei Fälle frühzeitiger Erkrankung des Centralnervensystems. (*a*) Multiple tuberöse Sklerose des Gehirns; (*b*) Microcefalia vera (Giacomini)." *Archiv. f. Psychiatrie*, Band 30, 1898.

Schüle. "Weiterer Beitrag zur Hirn. Ruckenmarkssklerose." *Deutsches Arch. f. klin. Med.*, Band 8, p. 223.

Sherlock. "Anoia, a Form of Mental Deficiency." London, 1905. Baillière, Tindall & Cox.

Tredgold. "Amentia (Idiocy and Imbecility)." *Mott's Archives of Neurology*, vol. ii., p. 402.

Ugolotti. "Sclérose tubéreuse du cerveau associée à des altérations spéciales des autres organes." *Rivista di Patologia n. et m.*, 1904, vol. ix., fasc. 8. Ref. N. C.

Chapter 4.—Hypertrophy of the Cerebrum.

Synonyms.—Hypertrophic idiocy. Megalocephaly, macrocephaly.

Of these names hypertrophy of the cerebrum is in most common use, and, as I shall show presently, it fits histological findings: to the physiologist the name is not so apt, for whereas betterment of functional activity usually accompanies enlargement of an organ, the contrary is here the case.

It is wrong to describe tuberose sclerosis under the heading Hypertrophy of the Brain (*vide* "Quain's Dictionary of Medicine," last edition). By hypertrophy of the brain the majority of writers mean a specific disease in which simple, uniform enlargement of the organ, without definite lesion or cause, a state of cerebral gigantism, is the principal anatomical manifestation.

The correctness of including this condition in the category of sclerosis may be called in question. I do so, however, because certainly in the cases forming the basis of these remarks, induration was a pronounced feature, and I believe it has been so in all previously recorded cases. At the same time I recognise that the condition presents several important departures from the usual associations. In most forms of sclerosis shrinkage is an accompaniment, and for this shrinkage a proliferation of neuroglia or connective tissue is responsible; here, instead of shrinkage we meet expansion, and although, as I shall show, there is an undoubted proliferation of neuroglia, the disproportion between this and the induration is great. Further, the induration and the enlargement are general instead of local. And, touching causal factors, in the commoner forms of sclerosis an irritant, local or general, and a coincident destruction of nervous tissues have invariably been present, but in this disease we have no proof of the existence or action of an irritant and all the evidence is against any destruction of nerve tissue. It is evident, therefore, that we are face to face with an induration unique in kind, and obscure in origin.

Clinical Notes.

Case 1.—The first patient was a male, aged 29, who had been an inmate of Rainhill Asylum for fifteen years.

He was transferred from another asylum, and was without friends; hence no note concerning his family history or his childhood is recorded.

Being short in stature and having an immense head, a small trunk and delicate limbs poorly clad with muscles, he always presented a peculiar figure. All his movements were executed slowly, and he was readily fatigued, but there was no definite motor paralysis. So far as could be judged, cutaneous and special sensation were unimpaired. The commonly tested superficial and deep reflexes were normal. No disease was detected in the organs of the trunk. He did not suffer from epilepsy, and there was no evidence of inherited syphilis.

Mentally he was an imbecile with a low degree of intellect; his vocabulary was so limited and his concepts so simple that he was quite unable to converse; he did not know his name or his age, and was rarely heard to say more than "give me a mug of tea." He smiled when taken notice of and evidently enjoyed some sense of conceit. A creature of spiteful impulses, he derived pleasure from attempting to injure aged and apparently feeble fellow-inmates, but, unable to discriminate between those who could retaliate and those who could not, and being weak himself, he received as much as he gave. He was unclean in his habits and addicted to self-abuse.

Necropsy.

As megalocephaly and hydrocephaly are sometimes confused during life, and I chanced to have at hand a hydrocephalic skull from a man of about the same age, I first made a series of comparative measurements. The figures are of sufficient interest to set down.

	Megalocephaly.	Hydrocephaly.	Difference.
Circumference	59·5 cm.	64·3 cm.	– 48 mm.
Glabello-occipital length ..	19·6 ,,	20·8 ,,	– 12 ,,
Basi-bregmatic height	14·1 ,,	15·5 ,,	– 14 ,,
Vertical index	71·90 cm.	74·50 cm.	– 2·60 mm.
Cephalic index	77·1 cm.	82·6 cm.	– 5·5 mm.
Minimum frontal diameter ..	11·2 ,,	11·6 ,,	– 4 mm.
Stephanic diameter	14 ,,	14·5 ,,	– 5 ,,
Asterionic diameter	12·5 ,,	11 ,,	+ 15 ,,
Maximum transverse diameter ..	15·2 ,,	17·2 ,,	– 20 ,,
Frontal longitudinal arc.. ..	14·6 ,,	19 ,,	– 44 ,,
Parietal arc	16·4 ,,	20·5 ,,	– 41 ,,
Occipital arc	12·4 ,,	6·7 ,,	+ 57 ,,
Total arc	43·4 ,,	46·2 ,,	– 2·4 mm.
Vertical transverse arc, right ..	19·4 ,,	20 ,,	– 6 mm.
Vertical transverse arc, left ..	18·7 ,,	21 ,,	– 13 ,,
Total transverse arc	38·1 ,,	41 ,,	– 20 ,,
Basi—nasal length	10·1 ,,	10 ,,	+ 1 ,,
,, —alveolar length	9·4 ,,	9·4 ,,	0
Gnathic index	93·06 cm.	94 ,,	– 1·04 mm.
Asymmetry, frontal	R. + 4 mm.	0	
,, stephanic	R. + 10 mm.	0	
,, parietal	R. + 7 mm.	L. + 8 mm.	
,, asterionic	0	0	
Thickness of skull, frontal ..	10 mm.	3 mm.	+ 7 mm.
,, ,, occipital ..	12 ,,	5 ,,	+ 7 ,,

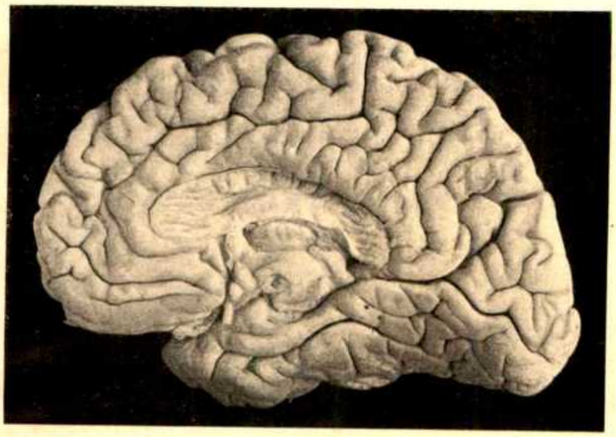


FIG. 5.

Hypertrophy of the Brain.

The inner surface of the right hemisphere from Case 1. In addition to the great size, the wealth of secondary sulci and the unusual distribution of parts in the region of the parieto-occipital fossa are noticeable.

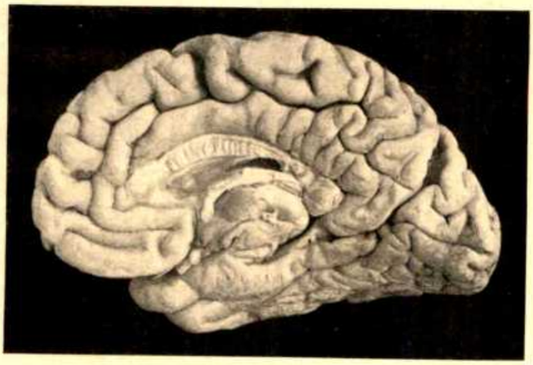


FIG. 6.

The inner surface of the right hemisphere of the brain (weight 1,350 gms.) from a normal female, for comparison with fig. 5.

These measurements do not diverge from what might have been expected, and they definitely prove that the brain enlargement in the disease we are considering is not associated with faulty growth of the cranial bones, as in hydrocephaly. The principal discrepancy affects the longitudinal arc; thus, although the frontal and parietal arcs were much greater in the hydrocephalic specimen, the collapse of the occipital bone, in the same skull, so reduced the occipital arc that the total measurements came to be approximately equal.

Passing to the cranial contents, the dura mater was normal, and there was no excess of cerebro-spinal fluid. The soft meninges were slightly increased in thickness and easy to strip, but free from opacity. The basilar artery appeared small, but the remaining trunks, and likewise the main veins, were normal in size and distribution, and I may mention here that microscopic examination disclosed no changes in them.

The brain was universally enlarged and scaled as follows:—

Encephalon	... 1,775	grms.
Right cerebral hemisphere (unstripped)	... 816	,,
,, ,, ,, (stripped)	... 775	,,
Left ,, ,, (unstripped)	... 768	,,
Cerebellum, pons and medulla ...	... 180	,,

The inequality in weight between the two cerebral hemispheres was not associated with any obvious asymmetry, the various lobes were well represented, in fact, the appearances of a large and uncommonly well-developed brain were offered. The surface colour was healthy, but handling proclaimed an abnormal and pronounced increase in consistence.

The form of the individual gyri was normal, and there was a complete absence of local atrophy on the one hand, and local hypertrophy on the other. On attempting to define the various named gyri and sulci, an arrangement of extraordinary complexity was found; indeed, in the whole of my experience, embracing a study of the morphology of several hundreds of brains intimately, and of several thousands casually, I have never seen one which presented such a wealth of secondary sulci, nor one in which guesswork had to be relied on so much in defining even the constant and better known sulci. But, since a full gyrological account will be of more interest to the anatomist than the pathologist, and will overburden the present article, I reserve details for a future anatomical paper. Here, in connection with the photographs reproduced, I will merely indicate that the fissure of Rolando has a remarkable course, and that the usual guide to its identification, the cingular incisure, is misplaced; that the arrangement of parts in the quadrate and cuneate lobules is much disturbed by an abnormal disposition of elements constituting the parieto-occipital fissure or fossa; and that many fissures usually represented by simple lines are extensively ramified.

In my first notes of this case I find the statement, "wherever a sulcal annectant gyrus can come to the surface it has done so, hence the complexity of gyral arrangement." This, however, is not correct, because although, for example, it is true that the great arcus intercuneatus and much of the superior Rolandic annectant are exposed, the cuneo-lingual annectants in the posterior calcarine fissure and many others remain submerged. Therefore that assumption has been abandoned, and now, after more careful inspection, I favour the belief that the remarkable sulcal complexity has developed on common mechanical lines; I would insist, however, on one fundamental difference between this and the normal brain, namely, that while the richly convoluted brain of the sane individual results from the restricted expansion of healthy substance containing a normal admixture of constituents, we deal here with the expansion of matter having qualitative defects, defects to be described presently under the heading "Microscopic Examination."

The cortex generally was increased in depth, but it is interesting that several of the usual naked eye, topical variations in depth and appearance were noticed; for instance, the precentral cortex was appreciably deeper than the postcentral; of the frontal cortex the prefrontal was shallower than the rest, and in the visual area the line of Gennari was plainly visible.

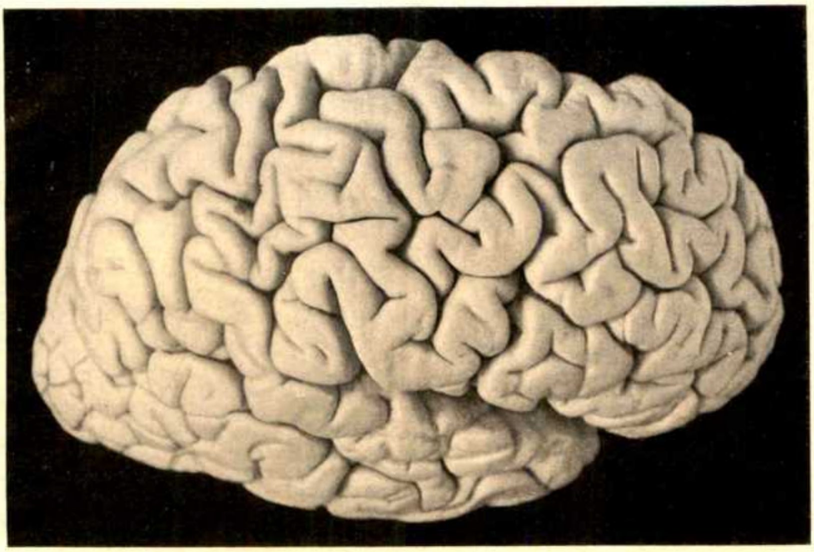


FIG. 7.

Hypertrophy of the Brain.

The outer surface of the right hemisphere, from Case 1.

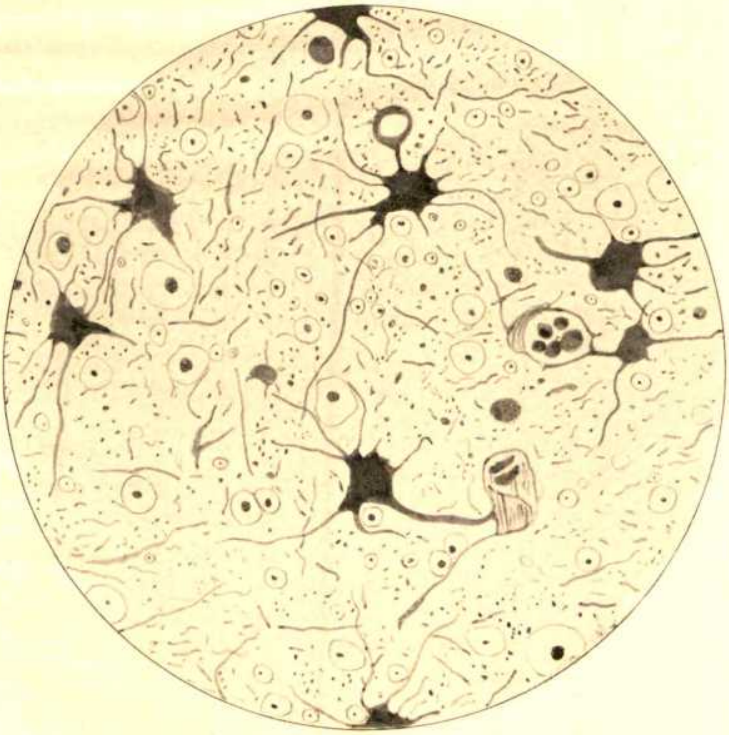


FIG. 8.

A reproduction of some of the large neuroglia cells present in the white substance of both cases of cerebral hypertrophy.

The white substance shared the general increase in bulk and looked healthy.

The lateral ventricles were not at all dilated; on the contrary, they were small in proportion to the size of the hemispheres; the ependyma was smooth. No abnormality of the basal nuclei could be seen. The corpus callosum was short, but stout. The anterior commissure was normal, the grey commissure smaller than usual. The septum lucidum, velum interpositum, choroid plexuses, and ventricular veins looked healthy.

The cerebellum, pons, medulla and spinal cord, were small in proportion to the cerebrum, but otherwise normal.

Examination of the trunk disclosed only the following points worth noting: a congenital malformation of the costal cartilage on the right side, the third and fourth being fused; universal plural adhesions, without thickening; œdema, congestion and some small gangrenous cavities in the lungs; old-standing mitral stenosis with much fibrous thickening of the cusps but little hypertrophy or dilation of the left ventricle or auricle; a small liver (945 grammes), congested and slightly cirrhosed, and congested spleen and kidneys.

Clinical Notes.

Case 2.—H. J. C., a male, aged 16, came from a private asylum, and again our record contains no reference to family or personal history.

Physically, no disability was apparent, and he was active in his movements.

A large but well-formed head and a bright expression suggested intelligence; on attempting to open a conversation, however, it was immediately discovered that he could neither speak nor understand the use of language; also, his general behaviour proclaimed a state of idiocy. He was mischievous; among other misdeeds he sometimes caused trouble by turning on gas and water taps, a product of imitation. When his wishes were not gratified, he displayed a vicious temper, and he was depraved in his habits.

It is remarkable that during the whole of his stay at Rainhill (five years) he did not have one epileptic fit, and yet status epilepticus—a series of forty-one seizures—was the cause of death. The convulsions took the following form: during the first ten seconds, drawing up of the legs, clenching of the fists, half flexion of the arms and twitching of the facial muscles, all bilateral, occurred in succession; then followed snapping movements and biting of the tongue. The concluding clonic stage was prolonged and the apnœic exhaustion severe, so much so, that at the end of the sixth fit the lungs were already œdematous. The usual remedies were administered without benefit.

Necropsy.

The state of nutrition and the muscular and osseous development were normal.

The head measured 22 inches in circumference, 8 inches in the greatest sagittal diameter and 6½ inches in the greatest bitemporal diameter. The forehead was high and the parietal bones expansive, suggestive of hydrocephalus. Placed anterior and internal to the parietal eminences were two additional symmetrical elevations as large as the parietal eminences. The interfrontal suture persisted. The calvarium was of normal thickness and the dura mater healthy. The pia arachnoid was opaque generally and thick, but not adherent. There was obvious congestion, but the vessels were normally distributed and had healthy walls.

For a boy of 16 years the brain was of great size. The encephalon weighed 1,515 grammes; the right hemisphere, unstripped, 672 grammes; stripped, 639 grammes; the left hemisphere, unstripped, 662 grammes; the cerebullum, pons and medulla, 145 grammes. The cerebral hemispheres were equal, and the representation of the lobes symmetrical and normal. The convolutions were perfectly formed, but the sulcal plan, without being abnormal, was complicated by many secondary ramifications, and various annectant gyri usually submerged appeared on the surface. The cortex and the white substance appeared to be healthy, but were distinctly increased in firmness. The ventricles were of normal size and the ependyma smooth. No fault was discovered in the commissures, basal nuclei, or remaining cerebral structures, and the same applies to the cerebellum, pons, medulla and spinal cord, though they were perhaps small in proportion to the cerebrum.

No change worthy of mention was found in the trunk.

Microscopic Examination.

The following account applies to both cases:—

(*a*) *Nerve Cells.*

Studying first the general lamination in sections taken from different parts and stained by the method of Nissl, it was astonishing not to find any positive abnormality. Whether taken from the frontal, parietal, temporal or occipital lobe, the sections showed an orderly columnar arrangement of cells, and both that disposition in layers and that representation of elements in the different layers known to characterise defined cerebral areas in the normal subject.

Going more into details I observed that the sectional outline of the cells, whether displayed by the method of Nissl, Golgi or Bielschowsky, was free from peculiarity, that the dendrons were normal in number and length, that in those cells which usually harbour chromophilic elements (Nissl bodies) such were visible, that when sections were silvered by the method of Bielschowsky neurofibrils appeared where they were expected, that there was no excess of pigment, and that none of those processless, pear-shaped, underdeveloped elements which we have seen in cases of idiocy were present.

Case of Macrocephalus.

	MM.	MM.
Layer 1	·278	·278
,, 2	·663	663
,, 3	·212	·743
,, 4	·236	
,, 5	·295	
Total		1·684 mm.

An Average Normal Case in Paraffin.

	MM.	MM.
Layer 1	·27713	·27713
,, 2	·83616	·83616
,, 3	·24117	·77875
,, 4	·23592	
,, 5	·30166	
Total		1·89204 mm.

(*b*) *Nerve Fibres.*

In sections stained by the method of Wolters-Kulschitzky I again saw nothing to associate with the defective mental state; indeed, had I been uninformed on the history of the sections and had I judged by the normal standard, I would have said that both the general supply and the arrangement of nerve fibres were those of an uncommonly well-developed brain.

(*c*) *Neuroglia.*

With the neuroglia it was otherwise, and yet the proliferation did not equal what reporters of other cases led me to expect. Nor, although the increase in glial elements no doubt contributed to the firmness of the cerebral substance, would I describe the condition as a gliosis. The microscopic picture was as follows:—

In the first or plexiform layer, a part wherein we find a decided excess of glial cells in another disease characterised by neuroglial proliferation, general paralysis of the insane, there was, it is true, some increase in the number of these elements, but it was not pronounced, nor did one see those great succulent cells so common in the disease named.

As is usual in other diseases, even in general paralysis, glial cells were not found in any number in the remaining cortical layers. In the white substance, however, it was different; here I have little hesitation in saying there was a true proliferation; moreover, on comparing the cells in these sections with the cells in sections from cases of advanced general paralysis, I observed very slight differences, structural or numerical. It did not matter how the sections were stained, whether by the method of Weigert, Bevan Lewis, Golgi or Bielschowsky, the cells stood out plainly, and I immediately classified them as homologous with the larger neuroglia cells of Bevan Lewis. As may be judged from the accompanying drawing the cells were of great size; they had many long, occasionally branched processes, and a stout vascular process ending in a sucker-like expansion on the vessel wall, and though the course pursued by the contained delicate, fibrillary elements seemed as usual to negative this, there could be no question that the processes were attached to and formed integral parts of the cell; the body protoplasm was granular and relatively copious, and enclosed a large, round or oval, deeply-stained nucleus. (Fig. 8.)

Such, then, was the prevalent type of neuroglia cell; in addition to these, cells of smaller size were noticed, but I am uncertain whether they were not large cells which had undergone retrograde changes; also, there was an abundance of what are called nuclei of the neuroglia.

(*d*) *Blood-Vessels.*

In certain parts, for instance, the occipital region, the blood-vessels stood out prominently because they were congested, but nowhere were vessels seen with thickened or diseased walls—another difference from the condition found in general paralysis.

REVIEW OF THE MANIFESTATIONS OF HYPERTROPHY OF THE CEREBRUM.

Although megalocephaly must be reckoned a rare disease, the number of cases recorded from time to time has now reached well over double figures. I do not pretend to have read all the reports, but I will insert a list here showing the weights of some of the brains described; it will serve a purpose in demonstrating that while the brains in my cases do not approach the record they are still of extraordinary weight.

Reporter.	Sex and Age of Patient.	Weight of Encephalon.
Walsem	Male, aged 21	2,850 grammes.
Simms	Male, aged (?)	2,400 ,,
Anton	Male, aged 20	2,055 ,,
Obersteiner	Male, aged 8	1,920 ,,
Brunet	Male, aged 18	1,780 ,,
Grant	Male, aged 43	1,869 ,,
Campbell	Male, aged 29	1,775 ,,
Bernardini	Female, aged (?)	1,755 ,,
Fletcher Beach	Male, aged 15	1,754 ,,
Brunet	Male, aged 17	1,632 ,,
Campbell	Male, aged 16	1,515 ,,
Fletcher Beach	Male, aged 16	1,500 ,,
	Average ..	1,892

Running over the clinical manifestations we find that males are more disposed to the disease than females, that most cases die before reaching the age of twenty-one years, that a condition of idiocy or imbecility is invariable, and that when the latter is the case, although something of what is said to them may be understood, the receptive process is greatly delayed, while if they have learned the use of language their vocabulary is restricted and their words uttered slowly, without emphasis and in a low tone. In about fifty per cent. of cases epilepsy is a complication, and that disease is a common cause of death; indeed, it may be correct to say that after passing a number of years free, or almost free, from epileptic attacks, life is terminated by a series of convulsions (*cf.* my second case). Although this observation has not always been made, the head seems to be enlarged at birth, and during childhood dulness, drowsiness and headache are suffered from. Further, such children exhibit that inability to keep the head erect which results from inefficiency of the cervical musculature; and in this connection let it be noted, that not only may the head be too heavy to balance, but the neck muscles may be abnormally weak, for in other directions there is proof of muscular inactivity; thus, many of these cases are reported to have been as slow in learning to walk as they have been imperfect in muscular development and incapable of enduring fatigue.

Turning now to the pathology of the condition, it seems that not unfrequently these cases are tainted with an hereditary neurosis, and have brothers or sisters afflicted with idiocy or some other form of mental weakness. There is also abundant proof that the morbid process is active during intra-uterine life; the large size of the head at birth, the presence of accessory parietal eminences and the persistence of the interfrontal suture which have been noticed, all signify abnormal cerebral growth at an early stage of development. Yet in searching for the cause further than this we cannot go. It is possible that the disease may rest on syphilis in the parents, but there is no certain evidence to that effect; indeed, it seems to me that not until a great many more observations are made and attention specially directed to obtaining correct family histories, shall we light on the right cause of this disease.

Next, regarding histogenesis, in few cases has a microscopic examination been carried out, and in fewer cases still has the brain passed through the hands of a competent microscopist, hence the histology of the condition is imperfectly reported. In particular, I notice a tendency to write loosely of the presence of inflammatory sequelæ in the brain and meninges; thus one observer makes capital of a superficial encephalitis (whatever that may mean) of the anterior third of the brain; another, recording a case in which death occurred in status epilepticus, apparently is unaware that capillary congestion and "myelocytosis" are simply characteristic of the disease which proved fatal; again, some thickening of the cranial bones and firm union of the sutures have been wrongly given as evidence of chronic inflammation of the brain. But, in my opinion, the case for the inflammatory process has been greatly overstated. To begin with, it is a fundamental truth that we do not get inflammation without vascular affection, and yet to the best of my judgment the blood-vessels in the brains I have examined have been remarkably free from disease; as blood-vessels are particularly numerous in the pia mater, therefore chronic inflammation always manifests itself in the soft meninges, but the thickening, opacity and adhesion of these membranes, which we regard as post-inflammatory signs, have not been present in my cases. Again, inflammation necessarily brings in its train malnutrition, destruction and decay, and yet here we find the principal and the most unstable elements actually represented in increased numbers. Lastly, while neuroglial proliferation is a product of both inflammation and decay, and it cannot be denied that this tissue is unduly abundant in megalocephaly, still, as I have pointed out, there is a great disproportion between the degree of the proliferation and the size and induration of the brain; moreover, neuroglia is a supporting element, and it is significant that it is excessive in the white substance only.

To me it seems that much more importance attaches to the proof we have that the enlargement of the brain is uniform and unattended by morphological imperfection. The architectural plan of the gyri and sulci may be unusual, but it is not bizarre; it is what we look for in the case of a person of high intellectual attainments, in accordance with the view that intellectual superiority runs hand in hand with advantage in cortical expansion. Inspection of known functional areas reveals nothing unexpected; for instance, on making a series of sections through the occipital lobe I observed that the area striata—visuo-sensory cortex—has a normal distribution; looking for giant "motor" cells, I find them where previous experience tells me they normally lie, and there only; indeed, the microscopic architecture shows no inconsistency in any part of the brain; in short, we have to deal with what seems to be a genuine cerebral hypertrophy.

In face of all these facts, the thought is forced upon us that this remarkable development of nerve cells, of nerve-fibres, and of cerebral nervous tissue in general, must be referred back to embryonic life, and tentatively I would suggest the influence of a developmental error. I would submit that there has been a deposit in excess of those embryonic elements which normally go to form the great system of cerebral neurones, a deposit quite out of proportion to standard requirements. Each primary specific centre (those for sight, smell, taste, touch and movement), each higher functional area, in fact the whole cerebral cortex, has been endowed with a wealth of fundamental elements far in excess of what is usual, and each element following a natural process has developed morphologically; but while development seems to have been good in so far as the central portion of the neurone is concerned, the same does not apply to the peripheral portion. To instance my meaning, let us take the motor area: for the perfect conveyance of impulses arising in the superabundant cortical motor elements, there should be a corresponding addition to the number of motor fibres in the motor tract; to the naked eye, however, the size of this tract in the medulla and in the spinal cord appears normal, and if, as is possible, there is an increase in number of the individual fibres without an increase in volume of the tract, a point which I have not attempted to prove, plainly there must be an insufficiency of investing myelin on each fibre, hence insulation must be imperfect. Of necessity, the same defect must obtain in the case of fibres for the conveyance of special sensory impressions; while among the multiplied neurones pertaining to higher functional areas, that coupling together, that association essential to the smooth working of the cerebral machine, as a whole has remained imperfect. The apparatus has been unwieldy, too complex to assume normal functional activity and impossible to educate. Although, to continue my instance, the cortex is endowed with good motor cells, their perfect excitation has been impossible, and the resulting movements sluggish; similarly, the process of education leading to the execution of skilled movements, an example of the use of higher function, has been out of the question, while in higher spheres still, complications and counteractions, clogging mental processes, have produced the clinical picture of idiocy or imbecility.

REFERENCES.

ANTON. "Wahre Hypertrophie des Gehirns mit Befunden an Thymus und Nebennieren." *Neurolog. Centralb.*, 1902, No. 21.

BÉLA SCHICK. "Zur Kenntniss der Hypertrophia Cerebri als Krankheitsbild im Kindesalter." *Jahrb. f. Kinderheilk*, vol. lvii.

BERNARDINI. "Ipertrofia cerebrale e idiotismo, osservazione clinicoanatomica." *Rivista sper. di Freniatria*, vol. xiii., 1897.

BRUNET. *Annales Médico-Psychologiques.* Paris, 1874. (Quoted by Ireland.)

CONSTANTIN TSIMINAKIS. "Zur Kenntniss der reinen Hypertrophie des Gehirns." *Arbeiten aus dem neurolog. Institut an der Wiener Universität.*, vol. ix., 1902.

COWIE GRANT. "Note on a Heavy Brain." *Lancet*, July 20, 1895.

CULERRE. "Idiotie avec hypertrophie du cerveau." *Archives de Neurologie*, 1887, T. xiii., No. 37.

FLETCHER BEACH. "A Clinical Lecture on Mental Deficiency in Children." *The Clinical Journal*, August 4, 1897.

IRELAND. "The Mental Affections of Children, Idiocy, Imbecility and Insanity." London, 1898. J. & A. Churchill.

TOMASCHEFSKI. "Zur Pathologie des Idiotismus." Inaug. Diss., St. Petersburg, 1892. (Ref. *Allg. Zeitschr. f. Psych.*, 1896.)

G. C. VAN WALSEM. "Ueber das Gewicht des schwersten bis jetzt beschriebenen Gehirns." *Neurolog. Centralb.*, 1899, No. 13.

CHAPTER 5.—CEREBRAL HEMIAGENESIS OR HEMISCLEROSIS.

A diminution in size affecting one hemisphere alone, equally distributed, associated with sclerosis but unaccompanied by disarrangement of the gyral and sulcal architecture, outlines the anatomical picture in the condition now to be considered.

It is not uncommon to meet with brains on the *post-mortem* table in which there is a disparity of more than 100 grammes between the weight of the two hemispheres, and in which the asymmetry is sufficiently decided to be noticeable with the eye; but in the disease before us the disparity in weight and the asymmetry are profound.

The condition is rare; I have seen it once only, and the records contain accounts of few cases which I would place in the same category. The Index Medicus mentions several papers entitled "Cerebral Hemiatrophy," but on reading these I have discovered that the "hemiatrophy" was a reduction in size due to some local defect, a restricted area of microgyria, sclerosis and contraction round an area of destruction consequent on arterial embolism or occlusion occurring in early life, and so on—not hemiatrophy strictly speaking, and a state quite distinct from that with which we are concerned. Finding that the name "hemiatrophy" had been loosely attached to various diseases, it became necessary to employ a term which would distinguish the present condition, hence the name "hemisclerosis"; this, though physically proper, may not meet the approval of the pathologist, who, as I shall indicate in commenting on the condition, may prefer the term "hemiagenesis."

The case which I shall relate has already formed the subject of a note by Dr. J. Wiglesworth in the *Liverpool Medical Chronicle;* I have restudied the brain from the histological aspect.

Clinical Notes.

The patient was a female, aged 16 years, an idiot and a sufferer from epilepsy.

The clinical report gives no information touching the family history and is very imperfect It is stated, however, that the

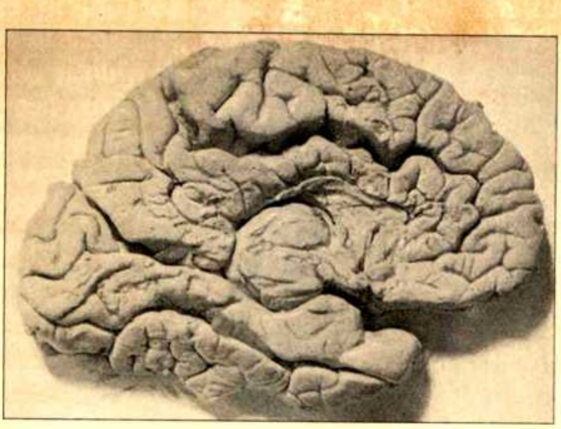


FIG. 9.

Cerebral Hemisclerosis.
Photograph of the inner surface of the left hemisphere.

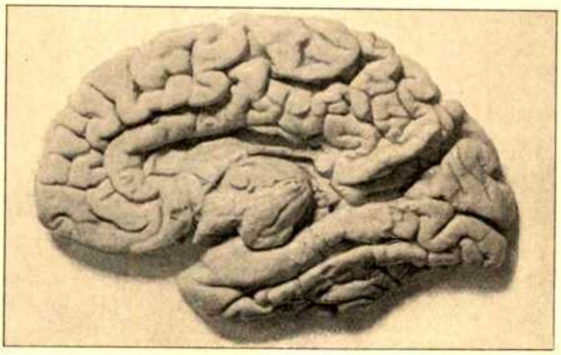


FIG. 10.

Photograph of the inner surface of the right hemisphere illustrating the asymmetry in the author's case. Although the sclerosed right hemisphere is so much the smaller it has preserved its form.

extremities of the left side were small and "shrivelled," and that talipes varus on the left side, and genu valgum on the right, made walking impossible, although the patient could stand. What power of movement she had in the wasted limbs is not mentioned, nor do the cutaneous and special senses seem to have been tested.

Necropsy.

The brain scaled as follows: Encephalon, 800 grammes; right cerebral hemisphere, unstripped, 218 grammes; stripped, 198 grammes; left cerebral hemisphere, unstripped, 417 grammes; stripped, 405 grammes; cerebellum, pons and medulla, 112 grammes.

The disparity in size and in weight between the cerebral hemispheres were features of dominant interest in the case. They were accompanied by many secondary changes in the cranium, but I need mention only that the right half of the head was obviously smaller than the left, and that this applied to the bones of the vault as well as to those forming the basal fossæ. In thickness and in density and as regards sutures, the bones were normal. So was the dura mater, save that a pale pink film,[1] composed of migratory and red blood cells, lined the vault on the right side. There were no abnormal adhesions between brain and dura. The cerebrospinal fluid was not markedly excessive. The pia-arachnoid membrane covering the right hemisphere was thick and opaque, and stripped in one sheet; that covering the left hemisphere was normal. The main arteries of the right side were smaller than those of the left. Anæmia was general.

Comparing the cerebral hemispheres (*vide* figs. 9 and 10), it was remarked that although the right was so greatly deficient in size it was not strikingly altered in form; every convolution was attenuated, every sulcus gaped and was shallow, all the basal nuclei were small, the white substance was wasted and the lateral ventricle dilated, but the general architecture was undisturbed, and it could not be said that any lobe or part in particular had suffered more than the rest. There was a striking difference in consistence, the lesser hemisphere throughout was greatly increased in firmness, cutting almost like costal cartilage; the same hemisphere was pale and pearly in appearance, and its cortex shallow and indistinctly striated. In all these respects the opposite hemisphere was approximately normal.

---

[1] This had no bearing on the principal change; such films are common in phthisis, the disease which caused death

Consequent on this cerebral asymmetry and the deficiency of cortical nerve cells and central nerve fibres, there were changes in related parts. The contralateral cerebellar hemisphere was distinctly reduced in size; the corresponding motor tract throughout the mesencephalon and spinal cord was wasted, and the lemniscus presented the attenuation of retrograde atrophy.[1]

The changes in the trunk included severe chronic tuberculosis of the lungs, secondary tuberculous ulceration of the intestine, and lardaceous degeneration of the liver, spleen and kidneys.

Microscopic Examination of Cerebral Cortex. (Fig. 11).

(*a*) *Nerve Cells.*

A careful examination of many pieces taken from corresponding parts of both hemispheres revealed uniform changes throughout the affected half. On the whole the cells preserved their columnar arrangement and the individual members their erect attitude, but the cortex was reduced in depth by quite two-fifths, and the lamination altered in the following manner.

The plexiform layer (1) was very thin.

The small pyramidal cells (2) were deficient in number.

The numerical deficiency fell even more heavily on the medium-sized and large external pyramidal cells (3 and 4), and the shallowness of the combined layers struck the eye immediately; moreover, few elements persisted here which might be described as normal; the majority were small and round, or oval, instead of pyramidal, their processes were stunted, they lacked spongioplasm and were achromophilous. Such cells recalled alike the neuroblasts seen in the developing brain, the stunted elements in cases of idiocy, and the atrophied cells in specific centres altered by interruption of incoming stimuli (cases of old-standing deafness or blindness, of tabes dorsalis, &c.).

---

[1] I investigated the changes in these parts very carefully, but do not describe them at length because they were an exact replica of what we have often seen in other cases of unilateral cerebral defect.

A few small cells remained to mark the position of the stellate layer (5).

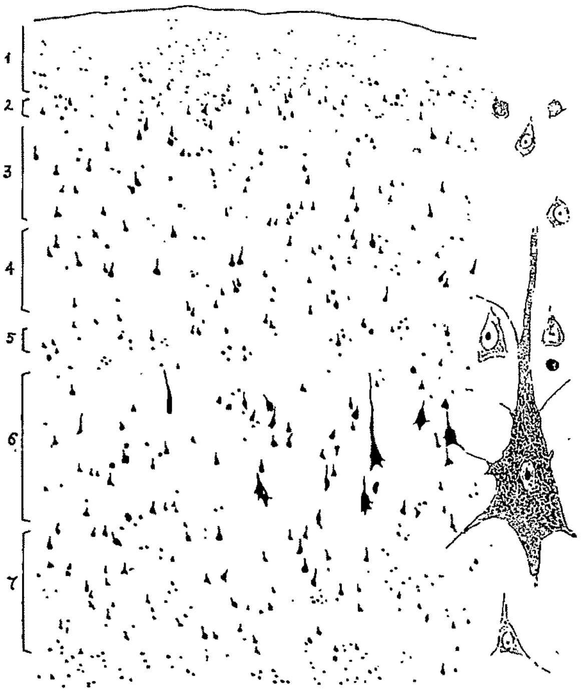


Fig. 11.

Camera lucida drawing of the cell lamination in the cortical motor centre for the leg in the diseased hemisphere. The cortex is shallow, the reduction in depth mainly affecting the suprastellate layers.

It is a remarkable and important feature that in cortex where the cells of the next layer—the internal layer of large pyramidal cells—are specialised, such cells persisted and showed but minor alterations. For instance, in the motor cortex, which I illustrate, the preservation of the giant cells was astonishing; their figure was attenuated, but their number good; the method of Nissl showed chromophilic elements diminished in size, but not true chromotolysis, and

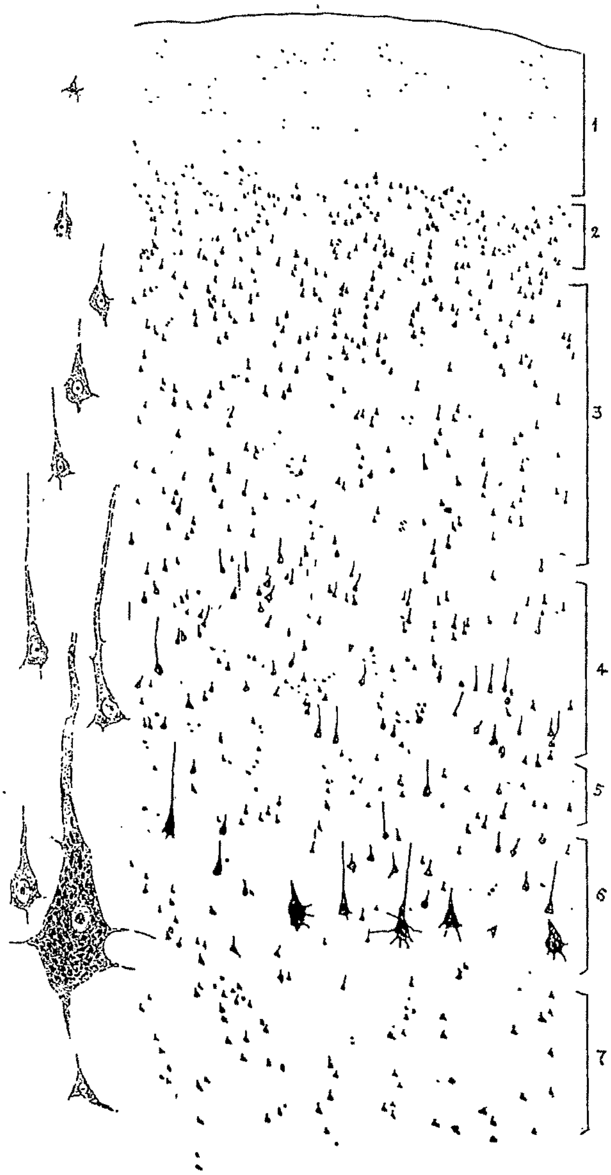


FIG. 12.

Camera lucida drawing of the cell lamination in the cortical motor centre for the leg in the normal hemisphere of the author's case of Hemisclerosis for comparison with Fig. 11.

the method of Bielschowsky revealed both a wealth of neurofibrils[1] and normal processes. Similarly in the visuo-sensory, in the postcentral and in the olfactory areas, areas wherein homonymous specialised cells are found, these among the cells of all other luminæ, save the fusiform layer, showed the best state of preservation.

The last layer, that of the fusiform cells, could be included with the layer just considered as preserving its constituents fairly intact.

Most of the points regarding cell lamination that I have mentioned are illustrated in the accompanying figures. The significance of the changes will be discussed in a later section.

(*b*) *The Blood-vessels.*

I searched many blood-vessels for signs of past inflammation, but the quest was negative. The vessels were not more numerous than usual, nor did thickening or over-cellularity of their walls cause them to stand prominent; also, signs of syphilitic endarteritis and of hyaline and other forms of degeneration were not in evidence.

(*c*) *The Nerve Fibres.*

Examining the nerve fibres as carefully as I examined the nerve cells, I again found changes in the lesser hemisphere as pronounced as they were universal.

The zonal layer in all regions, even in the motor area, where it is so prominent in the normal brain, was reduced to the palest of bands, made up solely of a few varicose fibrils. The supraradiary layer was correspondingly poor. A line of Baillarger was scarcely visible.[2] The radiations of Meynert, instead of being stout and composed of many large medullated fibres (I refer to the motor cortex) were attenuated, made up of coarse varicose fibres, and seldom strengthened by a normal evenly-medullated fibre.[1]

---

[1] The persistence of the neurofibrils is not easy to explain. In other conditions I have displayed them in cells quite denuded of Nissl granules. Evidently more information is needed regarding their nature and significance.

[2] In my "Histological Studies on the Localisation of Cerebral Function," I pointed out that the line of Baillarger lay on a level with the external large pyramidal cells, and that its representation varied proportionately to the representation of these cells. The condition of the line in this brain accords with that observation.

The interradiary spaces were almost empty, even in parts like the motor area, where in the normal state they are packed with fibres.

In short, a general loss of fibres was apparent, and I think it correct to summarise with the statement that the deficiency fell most gravely on the various systems of association fibres.

(*d*) *The Neuroglia.*

Large active neuroglia cells like those seen in the white substance of the normal brain were scarce; in all parts, however, there were numbers of the smaller variety of cell, as well as an abundance of so-called nuclei of the neuroglia, also the glial feltwork was uncommonly dense. The condition suggested a past glial proliferation to compensate the cell and fibre deficiency.

The origin of this condition is not easy to discover, and several speculations have been urged concerning its pathology; these we will briefly consider.

The weak suggestion has been advanced by one writer that it may be the outcome of unilateral narrowing of the carotid canal. No doubt narrowing of this canal will reduce the flow of blood along the middle and anterior cerebral arteries, and if it occur early in life may well interfere with the development of the parts of the brain nourished by these vessels; but surely coincident obstruction of the homolateral vertebral artery by narrowing of the foramen in the atlas, and over and above these constrictions, anastomotic imperfections, such as inelasticity of the Willisian arteries of communication, would be necessary to make the wasting of the hemisphere complete. There is no proof of any such obstructions, so that the suggestion need not be further pursued.

---

[1] It may seem incongruous that in the motor area, where the giant cells were preserved, the radiations of Meynert were attenuated. It will be remembered, however, that while these cells retained their shape they were reduced in size and showed internal alterations. And I have little doubt that if it had been possible to trace the axon of one of these cells, it would have been found attenuated and wanting in myelin. This supposition is consonant with an observation I made to the effect that the wasted pyramidal tract leading from the affected hemisphere retained a blue tint, instead of being decolorised, in Weigert-Pal specimens, and that on microscopic inspection the contained fibres, though not wholly amyelinic, were mainly composed of axis cylinder.

Of unilateral synostosis of the cranial bones preventing cerebral expansion there is likewise no evidence.

Injuries at birth are fruitful sources of brain lesions, and syphilis in parents or child are disease-producing factors always to be kept in view; here, however, we have no trace of the operation of either agent.

An attack of meningitis during infancy is more deserving of consideration, because we can readily believe that such an inflammation will not pass away without affecting the calibre, the elasticity, and the carrying capacity in general of the main arteries. This, notwithstanding there are several objections to meningitis as a cause of the condition of the case before us. In the first place, meningitis is not a disease which confines its attack to one hemisphere, and touching its effect on the main arteries, although in the majority of cases it attacks the base of the brain and involves the arterial trunks, it is as difficult to believe that it can confine its operations to the arteries proceeding to one hemisphere as it is to think of it spreading over one hemisphere and leaving the other intact. And yet other writers, particularly those of the German school, who lay stress on antecedent meningitis as a source of various changes which we see in the brains of idiots, might give causal importance to the thickening and opacity of the meninges noted in this case. To this I can only reply that the condition of the membranes did not suggest past acute inflammation; it reminded me of what I have observed repeatedly in the brains of the chronic insane, especially those afflicted with severe dementia, a condition of obscure origin, but certainly not the product of acute meningitis. The microscopic appearances, also, did not favour meningitis; in the cortex of this hemisphere we saw that, while cells and fibres were deficient, they were not arranged in a disorderly manner; the radiary fasciculi of fibres pointed straight towards the surface, and the cells stood erect and in column formation. This would not have been the case had meningitis pre-existed; in that event it would have been in accordance with our experience to have found the uppermost laminæ swept bare of cells and fibres, contracted and converted into connective tissue; moreover, from unequally distributed obliteration of cortical arterioles, patches of cortical destruction surrounded by contraction changes would have been seen in the deeper parts.

Having disposed of these preliminary speculations, we can now pass to the consideration of a problem which, in my opinion, it is of fundamental importance to solve. Did the condition exemplify atrophy of a part previously well developed, or was it the outcome of arrested growth? In my opinion it was the latter, and the following observations strengthened my belief. In form and in size the hemisphere resembled that of a full-term fœtus, but it was from the microscopic rather than the naked-eye appearances that I derived chief support for my contention. When studying the cell lamination we were struck with the fact that cells beneath the stellate layer, namely, the polymorphic and the internal large pyramidal cells, were preserved, whereas the cells above the stellate layer, in particular the medium-sized and large pyramidal cells, were very weakly represented, and to deficiencies in these layers we thought that the shallowness of the cortex was mainly due. Let us compare this with the state in the fœtal brain. As Dr. Bolton has proved, if the cortex of a fœtus be measured beside that of an adult, exactly those layers in the former will be shallow which were found to be so in this case; the corollary follows that the suprastellate pyramidal cells are the last to develop. Turning to deeper cells, it is well known that comparatively early in intrauterine life the specialised large pyramidal cells peculiar to certain areas—the cells, be it noted, which persisted in this case—may be recognised, and that the fusiform cells, likewise persisting in this case, are not much later in making their appearance. The resemblance, so striking from this standpoint, does not end here. From the writings of Flechsig, Kaes, Vulpius and others, who have studied the myelinisation of the cortex of the growing brain, we learn that the tangentially-directed fibres, the supposed association fibres, become invested with myelin considerably later than the vertically-placed projection fibres. A replica of this stage of development was seen in this brain; the radiary fasciculi, though deficient in myelin, were fairly prominent; the zonal layer, the line of Baillarger, and the association fibres, on the contrary, were barely recognisable, and this even in parts where normally they stand out with the greatest plainness. Furthermore, the amyelinic condition of the fibres composing the pyramidal tracts was a reproduction of the fœtal state.

While maintaining that all these microscopic revelations favour my thesis that the defect in this instance is ascribable to arrest of development, it does not escape my mind that some of these data may be seized upon by those who favour the view that the condition is the outcome of atrophy. In particular I recognise that an attempt may be made to explain the deficiency of stellate cells on the ground that, being highly organised elements and the last to develop, so they are the first to disappear in processes of degeneration. This is an axiom the truth of which cannot be gainsaid. Also, I will not deny that in cases of atrophy the change is dominant at the level in question; but in all my experience of cortical wasting, I have not yet met with a case in which other layers besides the suprastellate have not shared the change, in which, to be more precise, the specialised substellate pyramidal cells, the fusiform cells and other elements, have not exhibited pigmentation, swelling, disintegration, numerical deficiency, and many other signs of degeneration. In this manner, therefore, wasting differs from the condition in hand. Further, and this is an important point, the profound asymmetry of the cranium contraindicated atrophy of its contents as strongly as it suggested their restricted growth.

Accordingly, I submit that we have to deal here with a true case of arrest of development, that the original deposit of elements going to form the nerve cells and nerve fibres was not deficient, and that growth proceeded normally until just before, at, or soon after birth (the conformation of the hemisphere is preserved), but then ceased; that the elements subserving primary essential functions, being developed already, persisted; that the elements subserving higher evolutionary functions, being only partially developed, and therefore unstable, to a large extent, but not wholly, suffered decay, and that coincident with this decay the neuroglia proliferated, hence the sclerosis.

LIST OF REFERENCES.

BINSWANGER. "Ueber die Entstehung der in der Kindheit erworbenen halbs. Gehirnatrophie." Dissertation. Strassburg, 1875.

BOURNEVILLE. "Sclérose cérébrale hémisphérique: idiotie, hemiplégie droite et épilepsie consécutives." *Archives de neurologie*, 1897, Tome iii., No. 15.

COWAN. "Notes on a Case of Cerebral Hemiatrophy." *Jour. of Mental Science*, 1895, p. 420.

MARTINOTTI. "Atrofia di un emisfero cerebrale dell' emisfero del cerveletto del lato opposto." *Annali di Freniatria*, 1898, vol. viii., Fasc. 3.

SCHMAUS. "Diffuse Hirnsklerose." *Virchow's Archiv.*, Band 114, p. 154.

CHAPTER 6.—LOBAR AGENESIS WITH SCLEROSIS AND MICROGYRIA.

I will now pass on to another remarkable condition, which from the standpoint of pathogeny I would classify with cerebral hemiagenesis, because, in my opinion, it is likewise a manifestation of arrested development. Anatomically it differs from hemiagenesis in being bilaterally distributed; but it bears a resemblance to this condition inasmuch as the gyri of the affected lobes, though attenuated and sclerosed, are not puckered, and the plan of the sulci is not disarranged.

Lobar sclerosis and lobar microgyia may be suggested as better names for the condition. I must point out, however, that these names have been affixed indiscriminately to cases of lobar sclerosis resulting from intrauterine destructive lesions (really cases of porencephaly), to the contraction and induration occurring round an old and extensive patch of cerebral softening, to postmeningitic atrophy and sclerosis, always without regard to the exciting factor, where the sclerosis or microgyria is but a sequel of the primary disease. This is surely wrong, and, in my opinion, now that our knowledge of the anatomy of the brain in idiocy is becoming more stable, it will be better either to abandon the titular use of these names, or to reserve them for conditions (they must be very rare) in which the sclerosis or the microgyria, being the primary and essential feature, is deserving of capital mention.

I am fortunate in having three specimens with which to illustrate my remarks. Of the subjects two were females, aged 17 and 32 years at death respectively, and one a male aged 35 years at death. All suffered from epilepsy, and though their mental state, as observed in Rainhill Asylum, suggested idiocy, a history of the display of a certain amount of intelligence during childhood proves that they were not cases of congenital amentia. Since the anatomical picture in all the specimens is alike, it will not be necessary to describe more than one in detail. I choose that from the female, aged 32 years, because it was the last observed.

Clinical Notes.

The patient was admitted to Rainhill Asylum at the age of 16. The mother informed us that there were four other children in the family, all healthy, and that this girl had attended school and been apparently normal until the age of 12, when she began to suffer from epilepsy. This was the signal for a mental deterioration so rapid that three years later she was completely demented and in need of asylum restraint.

On admission to Rainhill she would have been classed as a case of congenital amentia had no details concerning her earlier life been forthcoming. She seemed to understand nothing of what was said to her, she took no interest in her surroundings, and either would not or could not speak. No physical defect was discovered except some weakness of the left arm, and this proved to be transitory.

The further course of the case was devoid of interest. In summary form the notes state that she was a petulant, degraded, coprophagic ament, and a sufferer from frequent and severe epileptic fits.

Necropsy.

Though short in stature the limbs and trunk were duly proportioned, well clothed with muscles and free from deformity. The fat covering was normal and the mammæ were fairly developed. There were no traces of congenital syphilis. The scalp was thickly covered with coarse, black, curly hair. In the shape of the head and face there was nothing peculiar, and in regard to cranial size the following measurements are illustrative:—

Circumference of head	55 cm.
Arc measurement from glabellum to occipital protuberance ..	33 ,,
Arc measurement from ear to ear	30 ,,
Greatest transverse diameter	14·3 cm.
Greatest antero-posterior diameter	18·5 ,,
Cephalic index	77 cm.

The calvarium was very thick, yielding average frontal and occipital measurements of 12 mm. and 11 mm. respectively; the increase in thickness mainly affected the inner table; the structure of the bone was fairly open, and the thickening seemed to be compensatory. The lines of the meningeal arteries, strange to remark, were not deep. A groove of normal depth and breadth accommodated the superior longitudinal sinus. The sutures were close, but not obliterated. Broad furrows, following the lines of the sutures, which we have learned to associate with syphilis, were not present.

The dura mater and its sinuses were normal. A slight excess of subdural fluid was noticed. The main arteries, carefully inspected, were only a little more delicate than usual. The pia covering those gyri to be noted presently as attenuated, was somewhat thickened and easily stripped, elsewhere it was normal.

The cerebrum exhibited remarkable bilateral alterations affecting the frontal and parietal lobes.

The Frontal Lobes.

The frontal lobes in each hemisphere and almost symmetrically were greatly diminished in size; viewed from above they stood apart from one another; from the side they looked pointed and the orbital face was tilted outwards. Excepting the precentral or ascending frontal gyrus, which was normal in each hemisphere, not one convolution on the

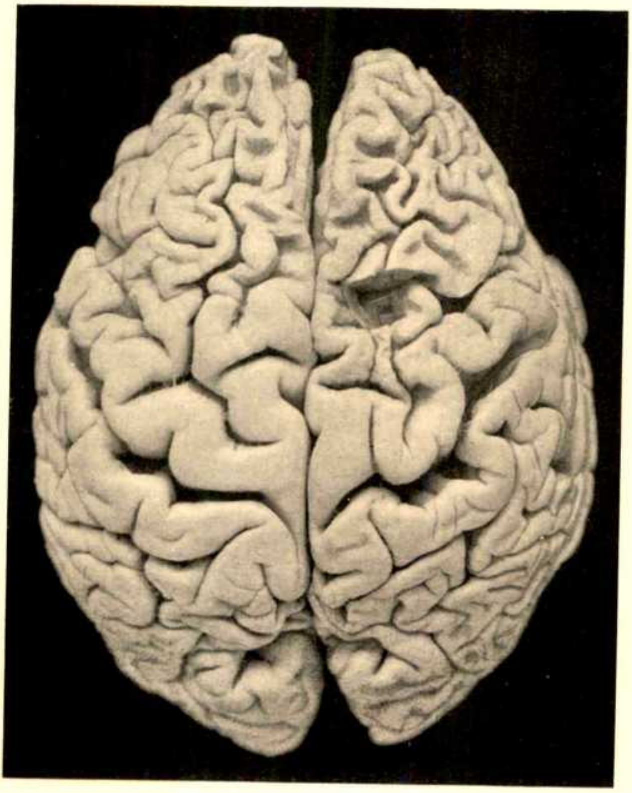
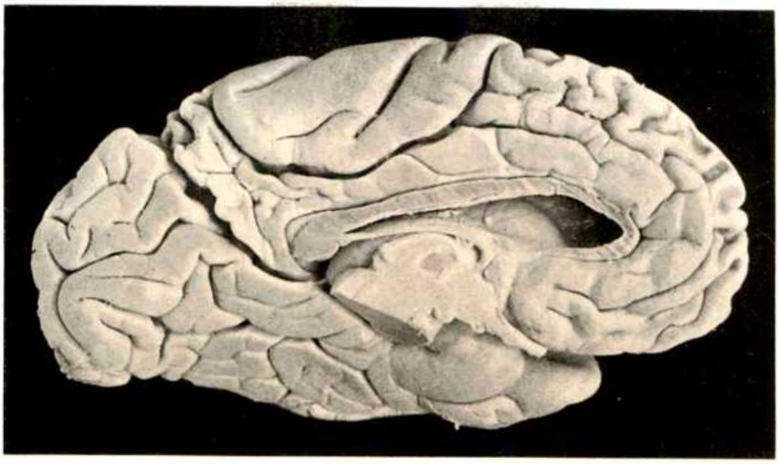
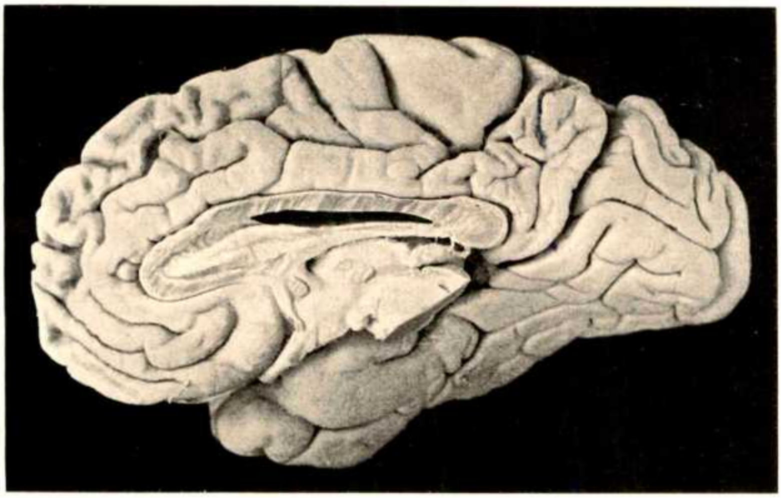


FIG. 13.

Lobar Agenesis with Sclerosis and Microgyria.

A view of the upper surface of the hemispheres in Case 1, showing the wasted frontal and parietal lobes.





FIGS. 14 AND 15.

Lobar Agenesis with Sclerosis and Microgyria.

The inner surface of the hemispheres in Case 1. Observe the bilateral and symmetrical attenuation of the gyrus marginalis and the precuneus; also, the large relative size of the oval lobule.

convex surface had escaped attenuation; the degree varied; those most changed were wormlike, pale brown and tough. those less affected, white and not so tough. On the mesial surface the marginal gyrus alone suffered, the oval lobule and the gyrus cinguli being normal. On the orbital surface all gyri, excepting those bounding the sulcus olfactorius, shared the change.

The Parietal Lobe.

Save the postcentral gyrus and its paracentral annexe which, like the precentral gyrus, were normal, the whole lobe was diminished in size and microgyrous. On the mesial surface the withering was very pronounced, but restricted in a most extraordinary manner to the precuneus and to that portion of the postcingular gyrus with which it stands connected. On the convex surface the wasting was less, and yet the superior parietal gyrus and the upper parts of the supramarginal, angular and posterior parietal gyri, were much below the normal size.

The Occipital Lobe.

The external occipital gyri were slightly attenuated, but those on the minor surface healthy.

The Temporal Lobe.

The temporal lobe stood out in that it was unaffected. The insular likewise remained untouched.

The Limbic Lobe.

The lobus pyriformis and all parts in the regio olfactiva were normal.

The corpus callosum, the fornix and the anterior and posterior commissures, were all slightly attenuated. The anterior horns of the lateral ventricles were dilated. The crura cerebri were small, but equal.

In the cerebellum some folia of the lobus clivi and others along the great horizontal fissure were wasted and unduly firm, apparently having undergone changes not unlike those in the cerebrum.

The heart was the only diseased organ in the trunk; it presented mitral stenosis (·45 inch), but evidently this was very old and had been naturally compensated, for the heart weighed only 243 grammes, and the size of the chambers relative to one another and the thickness of the walls was not greatly altered.

Microscopic Examination.

(*a*) *Nerve Cells.*

Portions of different gyri were examined, but to instance the changes I will take the condition of the cortex at the base of the superior frontal gyrus (regio precentralis intermedia of the author).

We have seen in our study of other brains that the cell laminæ most prone to decay are those which are the last to appear in embryonic growth, namely, the small, the middle sized, and the external large pyramidal cells (layers 2, 3 and 4). Exactly these laminæ were most deficient in this brain, and their weakness accounted for some thinning of the supraradiary plexus and the line of Baillarger to be noted presently. Of the internal large pyramidal cells many persisted, but they were all much reduced in size, and in addition so intensely chromophilous that even the nucleus was barely visible. The layer of fusiform cells, which is early to develop, which is constant throughout the animal series, and which is very resistive to decay, was the best preserved of all the layers. Lastly, the cell changes were evenly distributed, that is to say, the disorder and the patchy affection seen in inflammatory and vascular lesions were absent.

(*b*) *Nerve Fibres.*

The general fibre wealth of the cortex and of the medullary projection was greatly diminished, but of chief interest was the finding that the association system of fibres had suffered much more that the projection system. Thus, even in gyri which were most wasted, the radiations of Meynert, although attenuated to a considerable extent, were still prominent objects in the field, and they were strengthened by a few large medullated fibres, doubtless the axons of the remaining large pyramidal cells, and some large varicose fibres. The interradiary spaces, on the other hand, were comparatively empty; in particular they were quite bereft of those long, oblique or horizontal, large medullated fibres, which we look upon as associational or internuncial in function, and which are usually present in abundance in this situation.

As a feature of minor interest, the absence of a zonal layer, a wasted supraradiary layer, and a scarcely recognisable line of Baillarger were noticed, and, lastly, I will again emphasise the points that the radiary fasciculi stood erect, and that the signs of architectural disturbance were wanting.

(c) *Neuroglia.*

Neuroglia fibrils and so-called nuclei of the neuroglia were abundant in the diseased parts, and explain their firmness; but there were no large active neuroglia cells.

(d) *The Blood-vessels.*

The blood-vessels as seen in these sections showed no change calling for special remark.

Commentary.

In our search for enlightenment concerning the origin of the changes illustrated by these three brains, the first, indeed, the fundamental, point for consideration concerns distribution. In each specimen, with remarkable symmetry, precision and uniformity, the process is distributed over and restricted to the frontal and parietal lobes; in the former all gyri suffer, excepting the gyrus precentralis and some on the orbital surface; in the latter the postcentral gyrus alone escaped, and the precuneus is singled out for specially severe affection. To explain this distribution the series of causal factors from which we may choose includes vascular obstruction of different kinds, trauma, specific forms of inflammation and anomalies of growth and development.

The first factor, vascular obstruction, deserves careful consideration, because to the morbid anatomist the naked-eye appearance of the altered gyri at once suggests nutritional disturbance. Regarding this suggestion it is obvious that if there had been a reduced supply of nutriment, it must have been due to an affection of either arteries or veins, and if the former the cerebral changes should present an arterial distribution. But here at the very outset we meet an insuperable obstacle, the required agreement in not forthcoming. The atrophied gyri in the frontal lobe derive their blood supply partly from the anterior cerebral and partly from the middle cerebral artery, but not once in an examination of several hundred cases of softening from embolism and thrombosis of varying origin which have passed through my hands, have I seen a lesion having this distribution, even in one hemisphere, much less bilateral. Not only does it so happen that this part is one which is seldom the seat of a localised softening (a widespread softening from plugging of the anterior or the middle cerebral artery is of course not uncommon), but it is impossible to believe that the twigs to this field should be selected for occlusion and other branches of the same arteries left open.

Under the same premise the parietal defect is still more difficult to explain, because a part is wasted which draws its blood from all three cerebral arteries.

The microscopic appearances still further negative this suggestion. I have stated that there was no distortion of the general cortical architecture, that the cells preserved their columnar arrangement, and that the projection bundles of fibres stood erect, and, in my experience, gyri attenuated and sclerosed as a result of deficient blood supply, for instance, those on the borders of an old patch of softening, are never free from this distortion. For these reasons I regard arterial obstruction as an untenable hypothesis.

Turning to venous obstruction, there is one condition worth more than passing notice, thrombosis of the superior longitudinal sinus. In several cases of thrombosis of this sinus which have come under my observation, I have noticed that the congestion and the resulting multiple capillary hæmorrhage have favoured special situations, namely, the parietal and the frontal lobes on the convexity, and it has been interesting to see, presumably on account of the cross anastomosis along the veins of Trolard, that the two central gyri, just as in the cases under notice, escaped. Bearing these observations in mind, I can imagine that if a thrombosis of the superior longitudinal sinus were recovered from —it is usually fatal—and the obstruction persisted, or only imperfect recanalisation of the thrombus took place, it would exert an atrophic influence on the parts in which we are interested. But, unfortunately, I have no evidence to put forward to prove the occurrence of this thrombosis, my suggestion is purely speculative. The condition of the superior longitudinal sinus is not always noted at an autopsy, and it was overlooked in one of my cases; in the other two, however, I satisfied myself that the channel was apparently normal at death, whatever it may have been previously.

Touching trauma as an agent, all our evidence is negative. I suppose we may take it for granted that the chief manifestation of an injury is a rupture or thrombosis of blood-vessels; here there are no hæmorrhagic residua, and reasons have been given which render antecedent rupture of arteries unlikely.

Meningitis, encephalitis, and other inflammatory conditions, as possible causes of altered nutrition and subsequent atrophy, are all similarly disqualified by the remarkable distribution of the change.

Having found none of these hypotheses sufficient to explain the condition, we will lastly discuss the possible influence of a developmental defect, and now several very interesting points will be presented for consideration. One feature of great importance meets the eye when we view these brains from the aspects of physiology and comparative anatomy; selecting first the negative side of the picture, the side showing the parts which are preserved as against those diseased, we observe that the intact areas are precisely those we have recognised as designed for the control of movement and for the reception of the different sense impressions, in other words, for the government of those simple functions common to and used as a means to survival by all animals; thus, the visual area in the occipital lobe, the lobus pyriformis, representing the chief known centre for smell; the central gyri for the control of movement and for the registration of cutaneous sensations; the gyri of Heschl and the temporal gyri concerned with hearing, are all plump and healthy. Turning to the positive side, we notice that the parts destroyed are those which have been the last to appear in the progress of phylogenetic development, and which are elaborated for the government of those higher psychic functions and faculties possessed by man alone, and making him pre-eminent among terrestrial animals. In other words the specimens afford a pregnant illustration of a reduction towards the elementary state obtaining in the lower mammals.

This view of the brain is full of instruction. It seems to be a general physiological truth that structures and parts whose development, alike in the ontogenetic and in the phylogenetic sense, is deferred, are designed and specialised to subserve high evolutionary functions; likewise we may regard it as a natural law that the same structures and parts are penalised for their greatness by being the first to deteriorate, both in the normal process of dissolution and decay, and when the organism is subjected to an adverse and devitalising environment. In the case of the brain the frontal lobes, excluding the motor area, and the parietal lobes may be pointed to as being the last phylogenetic addition to the central nervous apparatus, and it is an established truth of ontogeny that they are the latest to acquire their fundamental nerve cell and nerve fibre constituents. On these grounds I maintain that the withering of these very parts in the specimens before us must be more than a coincidence. I submit that the wasting is not the product of any of the ordinary locally-acting morbid factors, but the manifestation of an unnaturally premature decay of parts constitutionally disposed to early dissolution, and at the same time specially susceptible to the effects of evil hereditary influence or any other adverse agent which may proclaim itself during the progress of either pre- or post-natal development. On this and no other ground that I can think of can we explain those features which puzzled us before, the bilateral, symmetrical and altogether remarkable distribution of the change,

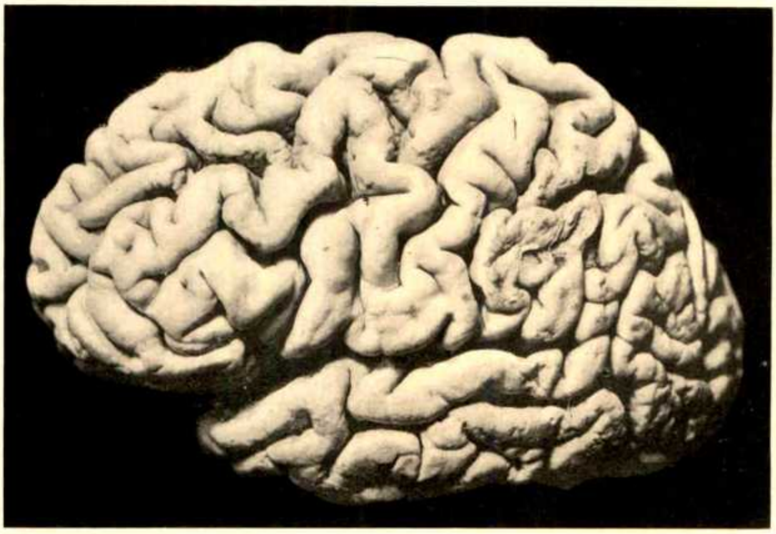


FIG. 16.

Cerebral Arterio Sclerosis.

A photograph of a cerebral hemisphere, showing puckered gyri in the frontal and parietal lobes.

the perfectly preserved architectural plan of the affected gyri and the well-ordered arrangement of cortical elements as seen through the microscope.

CHAPTER 7.—CEREBRAL ARTERIO-SCLEROSIS.

The morbid process now to be considered is chiefly of interest to the anatomist and histologist. In all parts of the brain, but especially on the outer surface, convolutions, or portions thereof, are irregularly picked out for attenuation and sclerosis; at the same time the surface is puckered by many minute pits and linear or radiate cicatrices, and withal the form of the gyri is not wholly lost.

As gyri in a similar state may be observed around patches of softening from arterial embolism and thrombosis, it must be explicitly stated that the present cases were independent of any gross lesion.

The condition is not common; although my experience has been among the insane, in whom cerebral lesions of all kinds are more frequent than in the sane, in a sequence of more than two thousand autopsies I have obtained only four specimens.

I will now enumerate briefly the leading features in each case.

Case 1.—A male, aged 24 years, who suffered from dementia with epilepsy. Both hemispheres were affected, the right more than the left. Analysing the distribution of the shrunken gyri, it was noticed that most lay within the area of supply of the middle cerebral artery, exceptions being some in the retrocalcarine region, pertaining to the posterior cerebral artery. The changes had not altered the plan of the fissures, and yet they must have been of old standing, because the cerebral disparity was accompanied by corresponding cranial asymmetery. The distribution suggested that the condition might have resulted from partial arterial occlusion such as arises in syphilis, notwithstanding that we could produce no evidence of syphilitic infection. Death was ascribed to phthisis.

Case 2.—A female, aged 80 years, who suffered from dementia with epilepsy. The withered gyri were scattered over the posterior and upper parts of the frontal lobes, the parietal lobes and precunei, and along the external parieto-occipital boundary. The arteries of the brain, indeed, of the whole body, were exceedingly atheromatous and calcareous, the basal nuclei showed *état criblé*, and there were a few small scars in the centrum ovale.

Case 3.—An obese woman, aged 64 years, who died of strangulated umbilical hernia. There was scarcely a convolution in either hemisphere which did not at some point show foci of cicatricial puckering. The associated reduction in size of the brain was so great that the weights are worth recording, encephalon, 970 grammes; right hemisphere, 402 grammes; left hemisphere, 402 grammes; cerebellum with pons and medulla, 138 grammes. The skull was very thick and the frontal bones rough internally. Calcification of arteries was general and extreme.

Case 4.—A female, aged 65 years, suffering from dementia. The brain was small (1,070 grammes), and, in addition to senile changes, presented withering of the gyri bordering the horizontal limb of the intraparietal sulcus and the second frontal sulcus in the right hemisphere. The case was unusual in that the change was unilateral. The subject was stout and well-nourished, the larger arteries were very calcareous, and the kidneys in a state of senile arterio-sclerotic fibrosis.

Commentary.

As three of these subjects were women, we may say that the female sex is disposed to the disease more than the male. Age also seems to bear an influence, because, although the man was but 24 years old, all the women had reached senility. The relation between the disease and an affection of the arteries is of greater importance. In the young subject the influence of syphilis could not be excluded, and in the remaining cases the arteries exhibited a degree of atheroma and calcification such as is not often seen even in cases of advanced senility; and, as I have already indicated, the distribution of the cerebral disfigurement favours the suggested relation to syphilis. At the same time, when we remember that rupture and thrombosis of such vessels are frequent in the aged, and particularly in the aged insane, it is singular that there was no gross lesion in any of these cases; and equally strange that in one case alone we found that dilation of the perivascular channels and those small scars in the central nuclei and white substance which we associate with degenerated vessels.

We will now enquire into the nature of the process, and here we shall be helped by the microscopic appearances.

Microscopic Examination.

Having made horizontal as well as transverse sections of many affected gyri, I have come to the conclusion that the change is attributable to a discrete occlusion of numerous

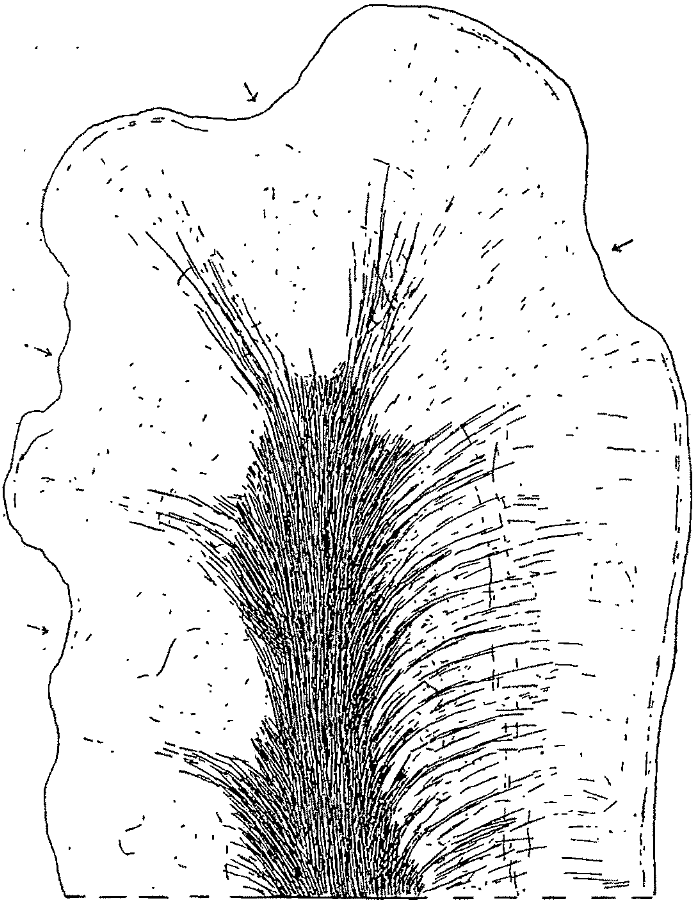


FIG. 17.

Cerebral Arterio-Sclerosis.

A semi-diagrammatic representation of a transverse section of one of the affected gyri, stained for nerve fibres. Along the margin are indentations which correspond with the pits seen before section. Beneath these the cortex is wholly denuded of nerve fibres and nerve cells and converted into glial tissue: elsewhere it is healthy. × 80.

cortical arterioles. First taking the appearances on transverse section, the cortex is divided into a series of healthy and diseased compartments; strips of substance entirely bereft of nerve fibres and cells, and composed of glial tissue only, alternate with strips wherein the nerve cells and fibres, although distorted by cicatrisation, are preserved; and it is interesting that each sclerosed strip lies immediately below one of the surface pits. Turning to the horizontal sections, the fields of sclerosis seen before as strips now appear as islets occupying about half a low power field of the microscope, most of them isolated and circular, but some oval and joined to neighbouring islets by a linear band of glial tissue. These islets are composed solely of fine glial fibres, and appear to be non-vascular; evidently by contraction they have drawn on the surface and caused the pitting which is a feature of the lesion. At the edge of each islet, in the adjoining healthy substance, one or more blood-vessels run, and also as a result of cicatrisation these are dilated. Elsewhere the vessels show hyaline degeneration and general senile changes. There is no indication of cyst formation, nor can hæmoglobin crystals be found.

To explain these microscopic appearances, we can only suppose that each strip or islet of sclerosis represents a field of substance originally supplied by a cortical arteriole now for many years occluded. Not to discover the occluded artery running down the centre of each strip is no proof against this origin, because time has converted the vessel into connective tissue, but it is hard to understand why some arteries should be selected for stoppage and others left patent.

In conclusion, gyral arterio-sclerosis with atrophy and pitting is a disease chiefly confined to aged dements with diseased arteries, it attacks the cortex in patches, and apparently is the outcome of a discrete occlusion of cortical arterioles.

CHAPTER 8.—COLLOID SCLEROSIS.

An affection of the blood-vessels is the principal factor in this form of sclerosis; glia-cell proliferation participates only to a minor extent. The condition is rare; for several

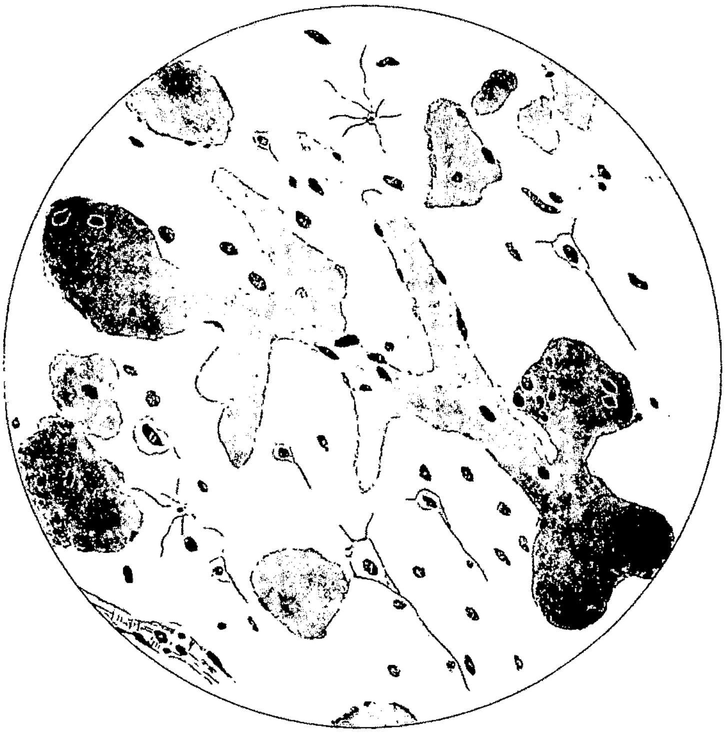


FIG. 18.

Colloid Sclerosis.

A microscopic drawing to indicate the size and shape of the colloid masses. The form of the main mass proclaims that it is an altered vessel. The drawing is taken from the level of the large external pyramidal cells. × 250.

years it was our routine practice at Rainhill to examine microscopically some part of the cerebral cortex in the case of every patient who died, and, with my later experience added, I have now notes of the inspection of more than 500 brains. In one case alone have the appearances I am about to describe been observed.

The subject was a man, aged 38 years. He presented the classical signs of general paralysis, and this combined with tuberculosis of the lungs to cause his death. As indicating general paralysis the brain was greatly wasted (weight of encephalon 1,162 grammes), the pia-arachnoid membrane was thick and opaque, the ventricles were dilated, and the ependyma was granular; and as the feature of special interest we found all the gray matter of the brain, including the cortex of both cerebrum and cerebellum, and the substance of the basal nuclei, greatly increased in firmness and abnormally yellow. Of course, it is the rule in general paralysis for the cortex to be unduly firm, but in this case the induration was profound, declaring itself not only to the fingers but to the knife.

The lungs provided examples of acute "pneumonic" phthisis, and, as proving the rapidity of its progression, there was no ulceration of the intestines.

The remaining organs were free from gross disease, and I must note specially that they were not lardaceous.

Microscopic Examination. (Fig. 18.)

In sections of the cortex and basal nuclei an abundance of large, semi-transparent, almost homogeneous masses immediately arrests attention; a few of these are cylindrical, but the majority are round, and for the latter 35 μ is an average diameter. Occasionally what appear to be concrete masses, giving a transverse measurement of about 180 μ, are seen. Many of the masses contain a few nuclei like those in vessel walls, but in other respects they are structureless. They present no concentric markings. They are almost transparent, and in the presence of most dyes retain their natural yellow tint; with methyl violet, however (in Weigert's stain for neuroglia), with hæmatoxylin, and with carmine, they stain intensely. With ordinary solutions of methyl violet a faint pink tint suffuses the intense violet colour, but, as proving that the reaction is not that of amyloid tissue, the masses neither turn brown with iodine nor blue with iodine and sulphuric acid. In the case of the basal nuclei the masses are more numerous in the caudate and lenticular bodies than in the optic thalami, and in the cortex they distinctly favour the walls and floors of sulci. The white matter is not deeply invaded.

Notwithstanding that this change cannot be shown in every section, there is no doubt that the masses are directly related to, indeed, represent, blood-vessels. On the question whether the process is a degeneration or an infiltration it is hard to pronounce an opinion, but, judging from general appearances, and particularly from the way in which the material follows each vessel in the form of a confluent mass, I prefer to think that we have to deal with an infiltration or a precipitation in and around the tissue elements rather than a degeneration. The change produces great thickening of the vessel wall, and here and there remarkable bulgings or buds (*vide* fig. 18), which are further curious in staining more intensely than the rest of the vessel. The isolated masses represented in the drawing are similarly thickened portions of surrounding vessels on partial section.

As to the nature of the material, I have little hesitation in declaring it to be colloid; it could only be confused with hyaline matter, but not if it be remembered that tissues altered by hyaline degeneration do not stain with methyl violet. Also I believe that the change is identical with that seen by Alzheimer in one case of general paralysis and in one of epilepsy.

REFERENCE.

ALZHEIMER. "Die Colloidenentartung des Gehirns." *Archiv. f. Psychiatrie*, 1898, p. 18.

CHAPTER 9.—GIANT CELL SCLEROSIS.

Perhaps more commonly than we imagine sclerosis manifests itself in a heterotopic anomaly of the nervous system in which a collection of giant nerve cells is an important

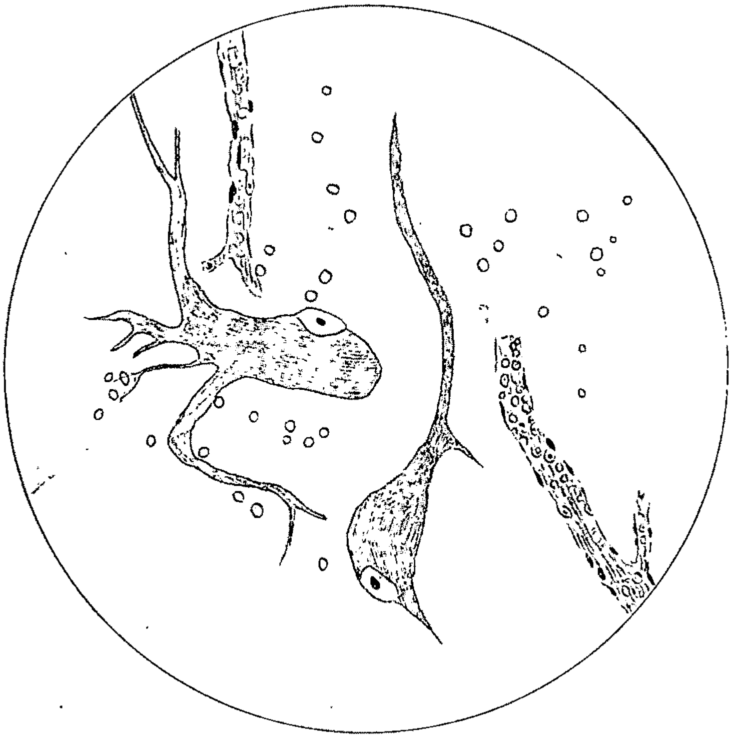


FIG. 19.

Giant Cell Sclerosis.

A camera lucida drawing showing two of the pallid giant nerve cells, with eccentric nuclei, which occupy the mass. Two capillaries and some nuclei of the neuroglia are included in the figure.

Stain, Thionin. Magnification × 300.

constituent. I have seen one case, an epileptic woman, aged 23 years, with various physical stigmata of degeneration, and a family history disposing to nervous disease and insanity.

In the second temporal convolution of the left hemisphere, four centimetres behind the tip of the lobe, imbedded in the white substance immediately below the cortex, was an indurated mass of the same colour as surrounding parts, and about the size of a hazel nut.

The microscopic sections I have prepared are occupied by numbers of giant multipolar cells, similar in shape and equal in size to those in the precentral gyrus (motor area), but their processes, which are long, point in all directions; their protoplasm is pallid, contains no Nissl bodies and is sometimes vacuolated, and the nucleus, small in proportion to the size of the cell, is frequently eccentric. Other nerve cells dotted about are small and polymorphous. The field is rich in nuclei of the neuroglia, and is interlaced by abundant fine capillaries. The overlying cortex appears to be healthy. (Fig. 19.)

I am conscious of transgression in including this condition in the category of sclerosis; still the giant cells are reminiscent of those seen in cases of tuberose sclerosis, and it was by the induration alone that the growth was detected. If it should not be accepted as a form of sclerosis, the name "glioma gangliocellulare," not so aptly applied to tuberose sclerosis, might meet the case.

CHAPTER 10.—OTHER FORMS OF SCLEROSIS.

There are several other forms of cerebral sclerosis, but being of common occurrence and having a known pathology they require only brief mention.

(*a*) *Sclerosis in General Paralysis of the Insane.*

We are familiar with the cerebral induration of general paralysis, and in the majority of cases do not err in ascribing it to the proliferation of glial tissue, and the universal thickening of vessel walls running in company with the neuronic degeneration characteristic of this disease.

(b) *Cerebral Sclerosis in Senility.*

As in general paralysis, so in senility, neuronic decay, vascular disease and glial proliferation combine to cause a general increase in the firmness of the brain.

In special cases, localised induration attended by extreme wasting of gyri occurs, and may be explained by atheromatous narrowing of the lumen of special arteries to the parts, whereby the blood supply, without being quite cut off, is much diminished, so aggravating the ordinary process of senile decay.

I have seen several cases in which the tips of the temporal lobes and the gyri on the convex face of the frontal lobe have been selected for affection in this manner.

(c) *Sclerosis of the Cornu Ammonis in Epilepsy.*

Although I have examined a number of brains from cases of epilepsy, I have found no support for the statement of early writers that sclerosis of the cornu ammonis is a common feature in this disease. Clothed by a dense, felted lamina medullaris externa, or zonal layer, and strengthened by radiating fasciculi of long and stout medullated nerve fibres, the cortex of the cornu ammonis, or gyrus dentatus, indeed, of the whole hippocampus, including the lobus pyriformis, is structurally firmer than other parts of the brain, and often feels much indurated, but not more often in epilepsy than in other diseases.

(d) *The Effects of Cerebral Softening and Hæmorrhage on Surrounding Parts.*

There is no doubt that the puckering and induration affecting gyri along the borders of a patch of ordinary embolic or thrombotic softening, or the parts around a hæmorrhage, are due to the severance and decay of internuncial neurons and the subsequent proliferation of neuroglia brought about by the arterial disturbance.

I have already mentioned that such cases have proved of great use to me in studying morbid conditions of the neuroglia, and there is no occasion to enter further into their histology.

(*e*) *Microgyria in Idiocy.*

In addition to the microgyrous conditions affecting idiots, described in foregoing sections, there is one which is very common, and though more frequent in the parietal and occipital lobes than elsewhere, it is not restricted to any particular part of the brain. The diseased area varies in extent, and is represented by a small nucleus of puckered and contracted, or wholly-destroyed, gyri, surrounded by others which are attenuated and sclerosed, but not deformed. There may be more than one lesion, and in any case considerable cerebral deformity and asymmetry results.

I refer specially to this condition in order that I may protest against the view that such areas are manifestations of some developmental error. I have seen many cases, and in all the distribution of the lesion and the constant signs of post-necrotic destruction in the centre of the area suggested a vascular origin, namely, embolism or thrombosis, at birth or soon after, of the artery leading to the part.

(*f*) *Cerebral Sclerosis in Syphilis.*

More or less widespread sclerosis of the brain has been reported in cases of congenital syphilis, but I myself have not seen an instance of the affection. In cases of gummatous subdural growths, however, arising in adults, I have observed infiltration and induration of the subjacent brain substance to a depth of several millimetres; and in other cases of syphilis, also in adults, I have noticed sclerosis and attenuation of gyri, particularly those of the frontal and temporal lobes, which I have ascribed to partial closure by syphilitic endarteritis of the supplying arteries, a condition which I have considered as akin to that sometimes seen in the brains of aged individuals with atheromatous vessels. Clinically, in such cases, the diagnosis of syphilitic pseudo-general paralysis has been sometimes made.

(*g*) *Disseminated Sclerosis.*

To complete the list of forms of sclerosis I have only to name disseminated sclerosis (sclérose en plaques) and military sclerosis, and mention that I have nothing to add to the descriptions of these conditions given by others.

THE CLINICAL HISTORY AND *POST-MORTEM* EXAMINATION OF FIVE CASES OF MYASTHENIA GRAVIS.

BY E. FARQUHAR BUZZARD, M.D., M.R.C.P.,

Assistant Physician to the National Hospital for the Paralysed and Epileptic, to the Royal Free Hospital, and to the Belgrave Hospital for Children.

With Five Figures.

IN a paper which was read before the Pathological Society of London I have already given a short summary of the histological findings in the five cases of myasthenia gravis which form the subject of this communication, but it appears to me desirable that a full account of these cases should be placed on record, inasmuch as they present features which are not only of pathological but of clinical interest.

The object of the present paper may therefore be regarded as two-fold. In the first place it will draw attention to some striking clinical phenomena occasionally occurring in this disease, and in the second place it will serve to propagate the view, which my experience has caused me to hold, that there are constant morbid changes associated with myasthenia gravis.

My best thanks are due to Dr. T. Buzzard, Dr. Ormerod, Dr. Head, and Dr. Wall, for their kind permission to make use of the clinical notes on the cases under their care.

Case 1.—A. J., aged 41, was admitted into the National Hospital under the care of Dr. T. Buzzard on November 3, 1903. His family history was too vague to be of importance.

His previous health had been good; but at 16 he contracted gonorrhœa, followed by no evidence of a sore or of secondary syphilitic symptoms. In 1892 he injured left eye and suffered from monocular diplopia for eight years. Moderate drinker of beer; no spirits. Occupation for twenty-one years had been that of a stone-grainer. Married nineteen years; fourteen children, of whom six died in infancy and one was stillborn.

PLATE I.

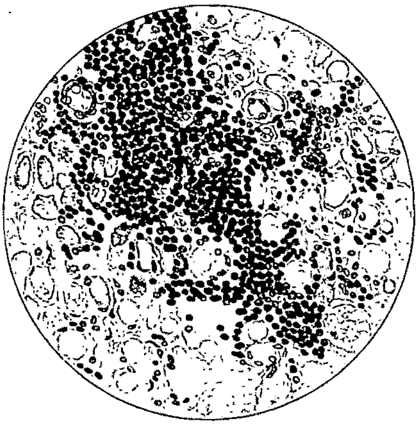


FIG. 1.

Section of inferior rectus muscle of Case 1, typical of lymphorrhages in ocular muscles.

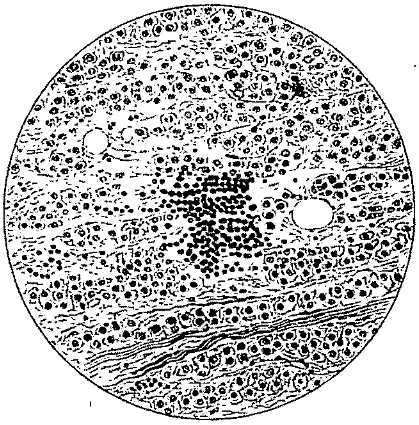


FIG. 2.

Section of adrenal gland in Case 4. A small lymphorrhage in the cortex of the gland.

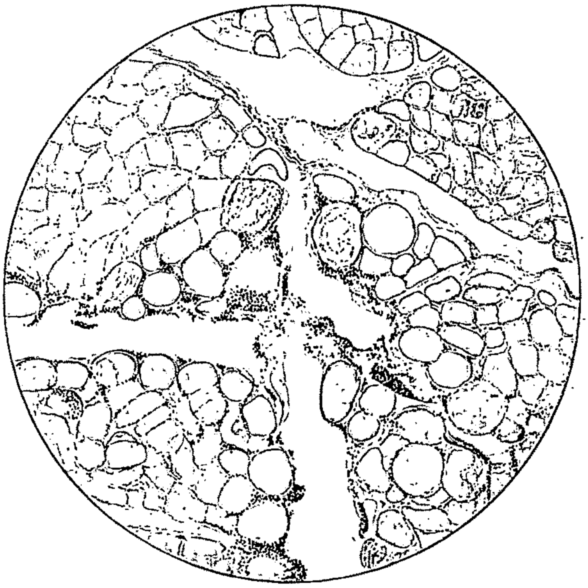


FIG. 3.

Section from triceps muscle of Case 5, showing marked changes in muscle fibres, some of which have been invaded by the cellular exudation.

Present illness.—For three or four years the patient had been obliged to lift his upper eyelids with his fingers when reading for any length of time. In other respects he had been well until March, 1902, when he noticed a feeling of heaviness in the right leg, which became easily tired. A month later he experienced an aching pain, first of all in the right foot, then in the left shoulder, and later all over the body, which would come on after he had been up for a couple of hours and remain until he turned in for the night. More recently he had been unable to walk more than fifty yards, after his work, without resting. For two or three months he had noticed a difficulty in swallowing, the bolus of food lying at the back of the throat and tending to get into the nasal passages. During the same period saliva would collect in his mouth and shortness of breath and difficulty in articulation would come on rapidly after a short conversation. At times his voice failed altogether, and his speech became unintelligible. He had lost two stone in weight during the previous year.

Condition on admission.—The patient was spare, but muscular, somewhat pale and tired-looking, and his movements sluggish and feeble. His forehead was almost constantly wrinkled in the effort to overcome the ptosis of both upper eyelids. As a witness he was bright and intelligent, answering questions readily and clearly. His appetite was good and he slept well.

With the exception that his heart sounds were somewhat feeble, all the internal viscera appeared to be healthy. There was no evidence of glandular enlargement in the mediastinum or elsewhere.

The examination of the blood showed 3,496,000 red corpuscles and 8,000 white corpuscles per cubic millimetre; Hæmoglobin 60 per cent. There were no abnormal forms of red or white cells, and the different varieties of the latter were present in the usual relative proportions.

Smell, taste, and hearing were perfect.

Sight $\frac{6}{6}$ with both eyes. Visual fields were normal. The fundus of each eye was healthy in appearance.

All ocular movements were very limited in range, the horizontal being restricted to about one-sixth and the vertical to about one-quarter inch.

The axes of the eyes were parallel and diplopia was not complained of. Pupillary reaction to light and accommodation was healthy. Marked ptosis was present on both sides. No nystagmus was observed.

Fifth nerve.—All movements of the jaw were performed with fair power. Opening and shutting the mouth was repeated 594 times before the patient was tired. On the other hand 45 momentary applications of a mild faradic current wore out the contractility of the left masseter. There was no alteration of sensibility in the skin of the face.

Seventh nerve.—All facial muscles were more feeble than normal, but the orbiculares palpebrarum and oris markedly so. The orbicularis palpebræ did not contract to a faradic current. The left zygomatics were exhausted by 50 applications.

The ninth and tenth nerves.—Deglutition was always difficult, even with the softest food, fragments of which were very apt to pass into the nose.

Liquids were swallowed more easily. Phonation tired rapidly, and might be absent altogether in the evening.

The palate was flaccid and moved very little, either on phonation or on reflex stimulation.

Twelfth nerve.—The tongue movements were fairly good, and it was not noticeably wasted. It was protruded ninty-one times before being tired.

The neck.—Patient could not raise his head from the pillow. When sitting up the head would fall forwards unless supported.

Upper extremities.—Movements performed by the muscles of the shoulder girdle were all feeble. The biceps was still more feeble, while extension of the forearm by the triceps could scarcely be carried out. Wrist movements were rather better, but both grasps were poor. All movements of the fingers could be made, but were easily resisted. There was no wasting of any muscle.

Trunk.—Patient could not sit up even with the help of his hands, but the abdominal muscles could be felt to contract in the effort. When in the sitting position the back assumed a round kyphosis, which could not be voluntarily straightened.

Respiration was partly thoracic and partly diaphragmatic, but shallow in type.

Lower extremities.—All muscles were well developed and their power much better than that of the muscles of the upper extremities. Extension of the right ankle was the weakest movement. The gait was good and no dragging of feet was noticed.

The co-ordination of all limbs was normal, and the sense of position accurate. No tremors were observed. Romberg's sign was not present.

Sensory system.—Aching pains in various parts were complained of, especially after movement. Attacks of sharp pain occasionally affected the inner side of his arms and his back. There was relatively diminished sensibility to light touches and to pricks over the ulnar aspects of the arms.

Reflexes.—The Jaw-jerk was present. Triceps and supinator-jerks were not elicited. Knee- and ankle-jerks were brisk. Epigastric, hypogastric and cremasteric reflexes were present. Plantar reflexes were of flexor type. The knee-jerk was not exhausted by 300 consecutive stimulations.

Electrical reactions.—To galvanism all muscles reacted normally. To faradism the following muscles reacted well at first, but were exhausted by varying numbers of applications.

Left sternomastoid	46	Right 50.
,, trapezius	14	,, 14.
,, supraspinatus	16	,, 31.
,, deltoid	52	,, 56.
,, extensor digitorum	114	,, 96.

January 29, 1904.—The patient has been suffering greatly with shooting pains, chiefly in the lower extremities, and from one or two attacks of respiratory failure, in which he was markedly cyanosed. The right knee-jerk is now much diminished and exhausted by six taps. The left is exhausted by twelve taps.

January 31.—Much weaker lately. Yesterday the temperature rose to 102° without obvious cause. Occasional profuse sweats are noticed.

The special senses are unaffected.

The patient has still marked ptosis which varies considerably, and is generally more marked in the evening than in the morning. There are practically no vertical or outward movements of the eyeballs, and inward movements are very limited. A week ago the only movement possible was a slight degree of conjugate movement to the left. Pupils react well. Chewing food causes rapid exhaustion of the jaw muscles. All facial movements are very feeble. He cannot fully close the eyes or whistle. The face is very expressionless. In smiling the mouth is merely elongated and its angles slightly raised. The muscles do not appear to be wasted. Fluid tends to run away from the corners of the mouth, and solid food collects between the teeth and cheeks. Regurgitation of fluids through the nostrils takes place occasionally. Swallowing of solids on some days is very difficult, and attended by choking and coughing. Articulation and phonation are easily exhausted.

The tongue is large and flabby, and all movements are readily tired on repetition.

There is no wasting of the muscles of the extremities, but they are flabby and toneless.

Movements of the neck are fairly good, but he is unable to hold his head erect for any length of time when sitting up. Patient cannot extend the upper limbs in front of him for more than a minute, and the left fails before the right. The grasps are feeble, but the relative strength of the movements varies considerably from day to day.

Respiration is chiefly diaphragmatic, and during the attacks of dyspnœa from which the patient occasionally suffers, the thoracic movements are very feeble and shallow. The act of coughing is poorly performed. No special weakness of abdominal or spinal muscles is observed.

Patient cannot raise either extended leg from the bed, nor can he extend the knees against gravity. Other movements of the lower limbs are relatively stronger. There is no inco-ordination, tremor or fibrillation observed in any limb.

All voluntary muscles are easily exhausted by rapid repetition of action, and there is analogous exhaustion of many of the weaker muscles by rapidly repeated faradic shocks. For instance, the biceps on each side is completely exhausted by sixty shocks, and the triceps by thirty stimulations. On the other hand, the pectoralis major was not completely put out of action by one hundred faradic shocks. Even when the muscles are completely inactive to faradism they respond briskly to galvanism. An effort to exhaust the muscles by direct mechanical stimuli was not successful.

Subjective sensations are divided by the patient into three classes: (1) Dull aching pains, chiefly in shoulders and back of neck; (2) a general tired feeling and a sensation of tightness and stiffness about the knees; (3) shooting pains of a sharp stinging character which dart from place to place in the lower limbs.

On testing there is considerable relative anæsthesia and analgesia along the ulnar borders of the arms extending over the ulnar half of each little finger. On the trunk the sensory loss involves a definite area between the second costal cartilage and a line about 1 in. below the nipples. There is also a slight degree of analgesia on the peripheral parts of both lower extremities. Muscle pain sense is also reduced in the legs, but there is no loss of sense of position in any limb.

Reflexes are in the condition mentioned in the previous notes.

The sphincters are unaffected.

April 11.—His condition remained unaltered until about ten day sago, when it became considerably worse. On some days the patient has scarcely been able to swallow at all, even his breakfast, and respiration has been difficult and associated with cough. Only a little pale mucus is expectorated after great effort, owing to the feeble nature of the cough. No abnormal physical signs are detected in the lungs. The urine is normal. The ptosis and ophthalmoplegia are much as before, but the pupils are smaller and react rather sluggishly to light. Mastication and jaw movements are very poor; towards evening the lower jaw drops, and has to be supported by the hands during conversation. There is some anæsthesia and analgesia over the central part of the face, including the nose.

Articulation and phonation are feeble.

The arms are much more weak; the patient can extend neither in front of him. Full closure of the fist is impossible. Abdominal muscles are definitely affected. The lower limbs are much as before, but more easily exhausted.

Subjective sensations remain as before, except that the shooting pains are more severe.

Analgesia is more intense and extensive than at the time of the last note. The ulnar half of each arm and both little and ring fingers are affected. On the trunk there is almost complete analgesia between the second costal cartilage and a level about an inch above the umbilicus. Analgesia is also more extensive on the lower limbs.

There is complete loss of sensibility to cotton wool on the whole of the upper extremities excepting a narrow strip along the radial border and thumb. The same condition obtains from the second costal cartilage to the umbilicus, and to a less extent below that level.

On the legs there is almost complete loss to the same test, but this fades off above the knees. The reflexes are as before, except that the abdominal and epigastric are not now obtained.

The fingers are now pointed and the skin over them thin, shiny and glossy.

The patient grew worse and had several attacks of respiratory failure, in one of which he died on April 26, 1904.

The autopsy was performed ten hours after death, when *rigor mortis* was well marked. The body was poorly nourished, but not emaciated. Nothing abnormal to the naked eye was observed in the course of removing the brain, spinal cord, and various nerves and muscles. The left eyeball was removed with the optic nerve and ocular muscles attached.

The thoracic organs were healthy in appearance, and only slight traces of the thymus gland could be detected in the fatty tissue of the superior mediastinum. The thyroid gland was well developed. The spleen was natural except for the presence of two small calcareous lumps in its substance. The pancreas, liver and kidneys were normal. The adrenal bodies were not obviously diseased. There were no enlarged peritoneal glands. Both scrotal sacs contained hydroceles with clear fluid—the testicles were free from disease.

MICROSCOPICAL.

Ocular muscles.—The muscles attached to the left eyeball were hardened in formalin and cut, some in paraffin, but the majority in celloidin, which gave much the better results. Longitudinal and transverse sections were made of each muscle. Most of the sections were stained by hæmatoxylin and eosin, but a few were stained by the Weigert-Pal method. It is unnecessary to detail the changes found in each muscle, as they were more or less the same, and the inferior rectus may be taken as a type of the others. Transverse sections across the body of this muscle, stained by the logwood-eosin method, presented unusual appearances, even with a low power, owing to the presence of three or four darkly-stained clumps of cells. These clumps were of various sizes, the largest measuring about ·2 mm. by ·15 mm. Under higher powers it was seen that these unusual appearances were due to the presence of a large number of cells, indistinguishable from the ordinary lymphocytes of the blood, lying scattered between the muscle fibres, nerve bundles and blood-vessels. Since these cell-clumps are characteristic and present in many parts of the body in this disease, I have proposed, for the sake of brevity, to denote them by the term "lymphorrhage." This name is chosen for two reasons. In the first place the cells are, as has been said above, indistinguishable from lymphocytes by their size, and by the fact that the nucleus is generally round or slightly oval and surrounded by an extremely narrow belt of protoplasm. In the second place they have infiltrated the tissues in the same way as red blood corpuscles do in the case of a capillary hæmorrhage, and are not, as a rule, associated with obvious changes in the surrounding structures, inflammatory or otherwise (Plate I., fig. 1).

In longitudinal sections similar lymphorrhages were met with, and some of these measured as much as ·3 mm. in length. Here, again, the cells were seen to lie in the spaces between the muscle fibres and not to invade them.

Every now and then a capillary vessel was observed running through the cells, generally empty but occasionally containing red corpuscles. Passing from the consideration of these lymphorrhages the condition of the muscle fibres next claims our attention. A careful comparison of the fibres in this case was made with those in similarly prepared sections from a normal ocular muscle, with rather indefinite results owing to the great difficulty in forming an opinion. It may be stated generally that the large majority of fibres were of normal size and appearance, but that possibly there was a greater variety in calibre than is seen in the healthy muscle. There is certainly not any marked increase of nuclei nor proliferation of connective tissue.

No abnormality could be detected in the nerve bundles or in the blood-vessels.

The sections stained by the Weigert-Pal method gave a beautiful picture of nerve fibres apparently well myelinated as far as their muscular connections.

Cardiac muscle.—The preparations of cardiac muscle stained by logwood and eosin were of perfectly normal character and free from any lymphorrhages.

Skeletal muscles.—Portions of the biceps femoris, vastus internus, tibialis anticus, and triceps muscles, were hardened in formalin and cut in paraffin and celloidin. A large number of sections were stained by the logwood-eosin method and examined.

Some longitudinal sections of the vastus internus showed fairly numerous lymphorrhages differing little from those seen in the ocular muscles. Some of them, however, were rather more extensive, from ·5 to 1 mm. in length. The cells lay quite free between the fibres, and there was no evidence of reaction in the latter. The sections taken from the other muscles did not present lymphorrhages, but it is hardly necessary to say that this fact does not signify an absence of lymphorrhage from the muscles.

Here, again, it was obligatory to investigate carefully the muscle fibres. In longitudinal sections the transverse striation was generally well marked and the fibres appeared healthy. In transverse sections it was noticed that a certain proportion of the fibres differed from their neighbours in two respects. In a transverse section of a normal skeletal muscle the fibres present polygonal shapes and fit together in a mosaic-like pattern. In the sections under consideration a number of fibres were rendered conspicuous by their rounded and non-angular contour and sometimes by their greater size. The same fibres were often still further differentiated by the fact that their protoplasm was more homogeneous and of a paler and more yellow hue than that of their healthy neighbours. Such variations from the common type are occasionally met with in presumably normal muscles, but are not nearly so numerous as in the sections obtained from this case and, as we shall see later, from other cases of myasthenia gravis. In all other respects the muscles appeared to be healthy, and sections stained by Busch's method showed no evidence of fatty change.

Cranial nerves.—A portion of the left trigeminal nerve close to its origin from the pons was stained in Busch's fluid, teased and examined microscopically. Not a single degenerated fibre was found. A portion of the oculomotor nerve, close to its central origin, and one of its muscular branches were treated in the same way and with the same result.

The right trigeminal nerve was stained by Busch's method and cut in celloidin; the sections showed no degeneration.

The ventral half of the crural region of the brain-stem, including the outgoing third nerves, was also examined by the Busch method with negative results.

A muscular branch of the oculomotor nerve was stained by the Weigert-Pal method and the fibres presented a perfectly healthy appearance.

The left optic nerve was cut longitudinally and transversely, and sections of it stained by Weigert-Pal and by logwood and eosin. Nothing abnormal was detected.

Spinal nerves.—A short length of the left ulnar nerve, taken from the region of the elbow, was stained by Busch's method and teased. No evidence of degeneration was observed in any of the fibres.

Another portion of the same nerve was embedded in paraffin and stained with logwood and eosin, but nothing abnormal was seen in the sections.

A piece of the anterior crural nerve was stained by Busch's method and cut in celloidin. No degeneration was found. Transverse sections of the sciatic nerve were stained by logwood and eosin and appeared to be healthy in all respects.

Spinal nerve roots.—The first dorsal anterior and posterior roots of one side were teased in Busch's fluid, but no pathological changes were detected. The posterior root of the opposite side with the ganglion attached was cut and stained with logwood and eosin. The root fibres were healthy in appearance, but owing to faulty fixation I do not feel inclined to express any opinion on the condition of the ganglion cells. In this ganglion and in several others from the dorsal and lumbar regions there were present a few small collections of lymphocytes scattered between the cells. As far as I could judge they were similar to the lymphorrhages in other tissues, although generally smaller.

Central nervous system. — The Busch, Weigert-Pal, Nissl and logwood-eosin methods were employed for the examination of the central nervous system after it had been hardened in 5 per cent. formalin. The parts examined included the left motor cortex, the oculomotor region of the brain-stem, the pons, medulla and various levels of the cord. With the exception of a few recent capillary hæmorrhages in the gray matter of the cervical cord the results were entirely of a negative character. Not a suggestion of early tabetic changes could be detected by the Weigert-Pal or Busch methods and the ganglion cells generally were of healthy character, as portrayed by Nissl's stain.

Liver.—Sections of this organ showed small scattered patches of fatty degeneration. Here and there were seen a few collections of lymphocytic cells resembling the lymphorrhages seen in the muscles. These appeared to be associated in most cases with a small intralobular capillary and sometimes a few of the neighbouring hepatic cells had undergone degenerative changes. In other respects the sections appeared healthy.

Adrenal bodies.—Sections from these glands revealed perfectly normal structures except for the presence of a few lymphorrhages. One or two larger ones were situated in the medullary part of the gland and a few smaller ones in between the cell groups of the cortex. It was noticed that the neighbouring capillaries were often engorged.

Pancreas. — Microscopical examination of this organ revealed no abnormal features.

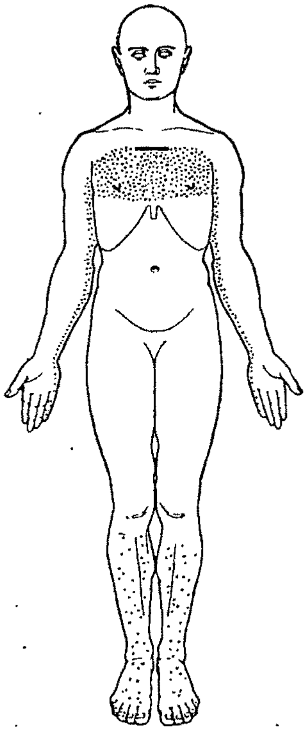
Kidneys.—As far as the glomeruli and tubules were concerned these organs seemed to be healthy. Now and then there appeared to be an unusual number of small round cells lying between the convoluted tubules. The aspect produced was different from that of the lymphorrhage in other organs because the cells were not lying together in rounded clumps but spread out between the tubules. It is difficult to express an opinion as to the abnormality of this appearance, and I prefer to leave the question open.

Testicle.—Sections of this organ presented no unnatural feature.

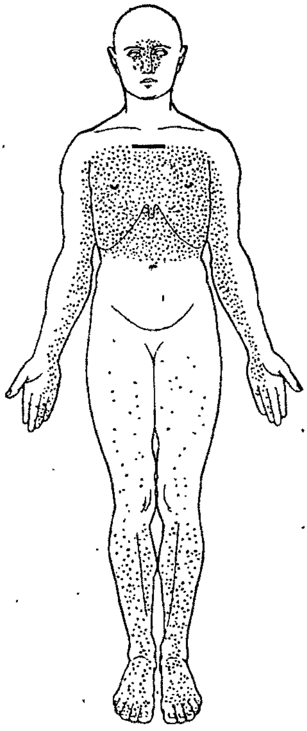
The thyroid gland, pituitary body and spleen. were all healthy as regards their histological appearance.

Thymus.—Report by Dr. Dudgeon. The fibrous tissue in which the scanty islets of thymus tissue are to be seen consists of very dense tissue without any nuclear marking in places. The cortex of the gland cannot be distinguished from the medulla. Many of the concentric corpuscles are very large and calcified in great part.

Case 2.—A Russian Polish woman, aged 28, was admitted to the National Hospital on October 8, 1902, under the care of Dr. Ormerod.



Analgesia, January 31, 1904.



Analgesia, April 11, 1904.

Family history.—Mother died of consumption. Father is alive and healthy. Five brothers and sisters are alive and healthy. There is no history of any similar condition in any relation.

Previous health.—Health was good with the exception of measles until the onset of present illness. Married eight years, but has never been pregnant. Husband is now 50 years of age and had children from his first wife. Patient's food has been of the ordinary character, and she has abstained from alcohol for some years.

Present illness.—Seven months ago patient began to feel pain in the shoulders, the arms and the legs and a sensation of pins and needles in the tips of her fingers. The pains were sharp and intermittent in character and were worse in wet weather. About the same time she complained of weakness in the arms, hands and legs. She continued for a month in this condition doing her housework, but always suffering and becoming weaker as the day went on.

Six months ago she experienced a sudden difficulty in talking during a conversation with a friend; she felt her mouth become stiff and her tongue immobile. About the same date she began to see double and to have a certain amount of dysphagia. The pains and weakness in the limbs continued and during the last few months her symptoms have become considerably worse. Lately she has found it difficult to prevent food coming back from her mouth through the lips, but has had no regurgitation through her nostrils.

Condition on admission. — Both upper eyelids droop and the lower lip is somewhat everted. Around the lips the skin is studded with beads of perspiration and the whole face is slightly flushed and pink in colour.

In smiling there is no elevation of the corners of the mouth, only a widening of the oral fissure and increased eversion of the lower lip. In voluntary movements of the face the action is slow and badly maintained. The patient is unable to whistle, but can blow out her cheeks. The attempt to close the eyes causes a movement of the feeblest character, especially on the right side. Elevation of the eyebrows is equally deficient.

The ocular movements are briskly and fully performed at first, but soon become partially exhausted. There is no nystagmus and at the present time no strabismus.

Opening and closing the jaws is well performed at first, but after twenty repetitions the observer can place his finger between the teeth and allow the patient to bite with impunity. The tongue movements are good at first attempts, but tire easily. The tongue itself is somewhat peculiar owing to its flabbiness, and to the presence of three linear grooves on its dorsal surface, one median and two about half way between the midline and the lateral edges.

The palate moves well on phonation and in response to stimulation, both sides being raised equally. Only on one occasion has the patient had any regurgitation of fluid through the nose.

Speech is normal. Articulation begins well, but soon tires. After frequent repetition the word "grub" becomes "grum," and by this time the patient is unable to blow out the cheeks without air escaping.

The pupils react to light and on accommodation, and this reaction was not exhausted by frequent stimulation or effort.

The muscles of the limbs exhibit diminished power generally, and this defect becomes much more marked on exertion, not only in the muscles exerted but in those of other parts. It was noticeable too that, after the limbs had been in a state of activity for a short time the ptosis of the eyelids would become more severe. The respiratory movements were of normal type and depth.

Sensation.—The patient complains of pain of two kinds: (1) general aching and stiffness, especially after exertion; (2) sharp intermittent pains as described above. On testing, cutaneous sensibility appeared to be perfect, except in a small area over the left shoulder, where there was relative anæsthesia and analgesia. It should be mentioned that the testing of sensation in this patient was unsatisfactory owing to her mental attitude.

Reflexes.—All deep reflexes were brisk and not exhausted by repeated stimulation. The corneal and abdominal reflexes were brisk, and plantar responses of the flexor type.

The sphincters performed their function in a normal manner.

Electrical.—The extensors of the left wrist were stimulated by repeated faradic shocks and completely exhausted in three minutes. At this moment very little voluntary power was left in the muscles, but a galvanic current produced a normal contraction with K.C.C. greater than A.C.C.

The left facial muscles were exhausted in ninety seconds, and on asking the patient to show her teeth no movement was obtained on that side of the face.

December 10, 1902.—General condition has improved, but strength varies much from day to day. The pains continue at times, and are not associated with any rise of temperature, but appear to coincide with increased weakness. There is now paresis of the right external rectus muscle and diplopia on looking to the right. The other movements are much as before.

Heart and lungs are healthy. There is no enlargement of thyroid, and the abdomen is normal on examination.

January 20, 1903.—Last night patient complained of great pain in her left leg. To-day she says that leg is weaker than the other, and this appears to be the case on examination.

There is no sensory loss.

There is dulness on percussion over the upper part of the sternum, and for about half an inch on either side of it. There is no tracheal tugging, and nothing to be felt in the episternal notch.

September 25.—Patient went to a convalescent home in June, and improved considerably during the first few weeks she was there. Five weeks ago she experienced a relapse, especially in swallowing and articulation. Since that time she has become gradually weaker and can now only swallow fluids and only talk for a very short time. She has suffered from sharp pains in the face, throat and neck. During the last ten days she has been unable to walk alone, and has had difficulty in breathing when lying down.

Physical examination reveals much the same as before, only the paresis is much more marked in the limbs and respiratory musculature. The knee-jerks can now be easily exhausted by repeated taps.

November 6.—The patient remained in practically the same condition, with variations, until to-day. This morning she seemed somewhat better than usual, but at 2 p.m. had a sudden attack of respiratory failure. When seen she was lying in bed with face, lips and extremities very cyanosed, and making feeble, shallow, gasping attempts at respiration. After artificial respiration had been performed, and a hypodermic injection of strychnine given, the patient recovered, but several attacks of the same character followed in rapid succession, and death took place at 3 p.m.

The autopsy was made four and a half hours after death, before *rigor mortis* had developed. The central nervous system, its meninges and enveloping bones, were all perfectly natural and healthy to the naked eye. The examination was limited owing to the wishes of the relatives, and only a few muscles and nerves were procured for microscopical examination.

The thoracic and abdominal organs were examined and removed through an abdominal incision. The heart and lungs were healthy. Lying in the superior mediastinum and loosely attached by its anterior surface to the posterior aspect of the manubrium sterni was a large thymus gland the vertical length of which measured 3½ ins. and the breadth about $2\frac{3}{4}$ ins. at its broadest level. Its shape was roughly that of a pear with a narrow stalk above and a bilobar gland below. The weight of the organ was 41 grammes, and its consistence was firm throughout. The glandular substance was surrounded by a small amount of fat which could be readily stripped off its surface.

The thyroid gland was felt in its usual situation and appeared to be natural in size, shape and consistence. It was not removed.

All the abdominal and pelvic organs were healthy except for a small cyst attached to the right ovary.

The various organs and tissues removed were hardened in 5 per cent. formalin.

After hardening the thymus was again examined and a section made through the middle of its broadest part. There was no evidence of necrosis or of malignant disease and the appearances suggested a simple hypertrophy of the gland.

MICROSCOPICAL.

Central nervous system. — The Busch, Weigert-Pal, Nissl and logwood-eosin methods were utilised for the purpose of examining sections from the motor cortex, brain-stem, medulla and spinal cord, but the results in all cases were negative so far as the discovery of any pathological changes was concerned.

Peripheral nerves.—Sections from the ulnar and sciatic nerves were stained by the Busch and Weigert-Pal methods with similar results.

Skeletal muscles.—Portions of the trapezius, deltoid, triceps and erector spinæ muscles were examined microscopically after embedding in paraffin or celloidin. Only a very few lymphorrhages were found and these were of small size. Numerous fibres, sometimes singly and sometimes in groups, were seen in transverse sections to be rounded in outline, often slightly swollen, and to react quite differently to protoplasmic staining reagents. With eosin or fuchsin the sarcoplasm assumed a pale and sometimes a rather bright yellow hue, the contrast to the pink colour of the normal fibres being a striking one. Sections stained by Busch's fluid also revealed the presence of these fibres which then assumed a darker brown colour than their neighbours.

Whether the stain used was eosin or Busch the homogeneity of the plasma was equally noticeable and longitudinal sections displayed the same features.

Cardiac muscle.—Sections from the left ventricular wall were stained with logwood and eosin and presented two points of interest. There were fairly numerous small lymphorrhages of a definite character, generally closely associated with capillary vessels. Except for the fact that the cells were not clumped so closely together, these generally resembled the lymphorrhages seen in other muscles. The second point to be noted was seen in a series of four sections which displayed a capillary blood-vessel engorged with blood and surrounded by a serous exudation of considerable extent. This exudation contained numerous lymphocytes but practically no polymorphonuclear cells and no red corpuscles.

Unstriped muscles. — Sections of the œsophageal wall showed no changes in the unstriped muscles.

Adrenal bodies. — Histologically these organs were healthy looking, except for the presence of lymphorrhages, which were numerous. They were found in the medullary part, in the cortex, and in the loose connective tissue of the capsule. One unusually large one in the lower layers of the cortex was traced through a number of serial sections and in each had a diameter of about half a millimetre. This one obscured and probably destroyed a considerable amount of the glandular structure, but many of the smaller ones caused little or no reaction in the neighbouring cells.

Liver.—A few sections of this organ stained by logwood and eosin showed the presence of a few lymphorrhages, the best marked of which lay in the centre of a lobule and measured not more than ·15 by ·25 of a millimetre. In other respects the sections were natural.

Kidney.—No lymphorrhages or other abnormal appearances were observed.

Spleen.—Healthy, the follicles being particularly well marked.

The pancreas, the pituitary body, and a bronchial gland were examined microscopically with negative results.

The thymus.—(Report by Dr. L. S. Dudgeon.) The gland presents a normal appearance under a low power of the microscope, and there is no evidence of fibrosis. The cortex and medulla are clearly differentiated, and the cellular constituents are of normal structure. There is, however, a point of great interest in the fact that, although the gland is enlarged and the structure is of normal appearance, yet there is a complete absence of eosinophile cells, such as are met with in the large thymus glands of children and young animals. These cells, which are often present in very large numbers along the septa and around the blood-vessels, are entirely wanting. The only condition in which I have found these cells to be few in number or absent, if the gland is enlarged, is in cases of hypertrophy of the adult gland. Many of the Hassall's corpuscles are degenerated. One or two of these contain calcareous masses which fill almost the entire corpuscle. Hyaline degeneration is also extremely well marked, and some of the cells have quite a clear centre surrounded by only a few cells at the periphery. Numerous hæmorrhages are present in the gland tissue and the small blood-vessels are very congested, a condition which is often associated with death from diseases accompanied by increase in intrathoracic pressure in young children.

Case 3.—N. S., aged 37, was admitted into the London Hospital, under the care of Dr. Head, on February 3, 1904.

Family history.—Her father is alive and healthy at over 60 years of age. Her mother is 59 years old and in good health except for varicose ulcers. Three sisters and two brothers are alive and well. Five brothers and sisters died before reaching 3 years of age; cause unknown. One brother died at 19, of diabetes. No history was obtained of any nervous disease in the family.

Previous health and habits.—Patient enjoyed good health, except for the ailments of childhood, until September, 1902, when she was laid up for a fortnight with inflammation of the lungs. She drinks about five small cups of tea daily, and never any alcohol.

Present illness.—At the beginning of October, 1903, the patient complained of some headache and vague pains, and noticed some "stiffness" of the eyelids on waking in the morning. During the following week she experienced double vision, and had to give up work. Drooping of the right eyelid was then noticed, as well as some difficulty in articulation. Improvement set in and continued until about a week after Christmas, when she appeared to catch cold. Articulation became more difficult, her lower jaw seemed to drop, and her head tended to fall forward. In spite of rest in bed her symptoms became worse, and towards the end of January dysphagia was observed.

Condition on admission.—The patient's complexion was rather sallow, but there was no abnormal pallor of the mucous membranes and no cyanosis. The facial aspect was peculiar owing to the bilateral ptosis and dropping of the lower jaw. Speech was a little indistinct, and in talking the lower jaw was supported by the patient's hand.

Vision was good and the optic discs and fundi healthy in appearance. The sense of smell was perfect.

The amount of ptosis varied; at times the upper eyelid covered the eyeball, but at others she was able to expose the lower half of the cornea. All external ocular movements were abolished, but the pupils were equal and reacted briskly to light and accommodation.

Sensation over the face was normal, but all movements of the jaw were markedly paretic.

The facial movements were so diminished as to render the physiognomy expressionless and flat. The orbiculares palpebrarum and oris were almost completely paralysed.

Hearing and taste were normal.

The palate moved quickly on phonation, but the movement was of small range and poorly maintained. Phonation was feeble and whispering in character.

The tongue showed no obvious atrophy, but was small and convex on the dorsal surface when protruded. After twelve attempts protrusion was so limited that the tip did not pass the teeth. A minute's rest sufficed to restore the original mobility.

The muscles of the trunk and limbs presented no wasting, no tremors, no fibrillary twitchings, and no spasticity.

The head fell forward unless supported, but, when support was given, lateral movements were freely performed. The sternomastoids responded briskly to the faradic current, but fifty short

PLATE II.

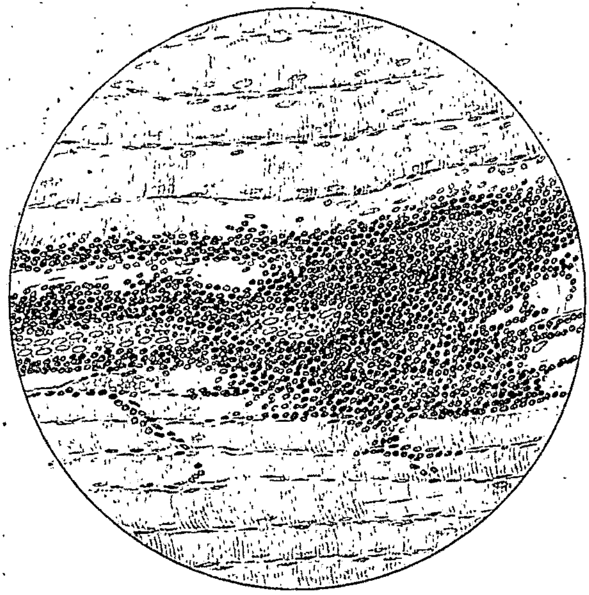


FIG. 1.

Section from the rectus abdominis muscle in Case 3, cut longitudinally and stained by logwood and eosin. Large focus of lymphocytes, healthy muscle fibres and, in the midst of the focus, a capillary vessel represented by a layer of endothelial cells.

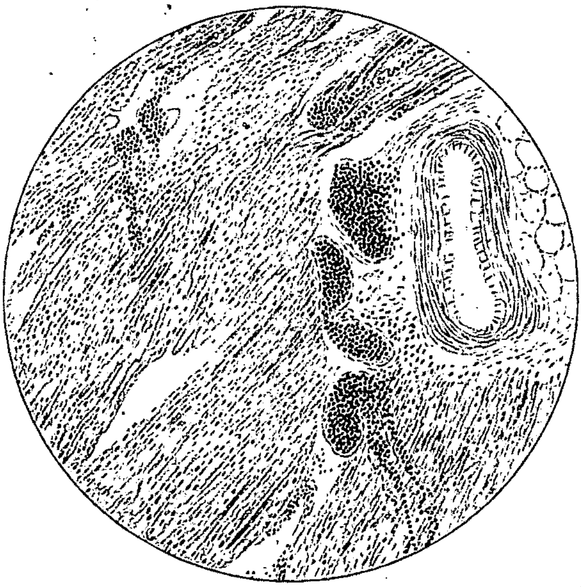


FIG. 2.

Section from cardiac muscle in Case 3, stained by logwood and eosin. Many of the cells are here contained in capillary vessels lined by endothelial cells.

applications were sufficient to abolish the response. The response to galvanism was not interfered with.

The limb movements were carried out with fair power and co-ordination. The patient had only recently noticed any undue fatigue after walking.

Sensation everywhere was perfectly natural.

The knee-jerks were equal and brisk, and there was no ankle clonus. Plantar stimulation gave flexor responses on both sides.

The spine, sphincters and respiratory system were normal.

The heart was normal, pulse good, moderate tension, arterial wall not felt.

March 18, 1904.—Patient seems much worse, and muscular weakness is more marked.

She had two choking fits yesterday and one in the night.

April 15.—(Note by Dr. Head.) On the whole she is better, and has been up on a couch. The speech is distinctly nasal, and she holds her jaw with her hand when talking in order to bring the lips together. She cannot completely lift the eyelids nor close the eyes firmly. Profound ocular paralysis is present, especially in the left eye, where upward, external, and downward movements are impossible. There is slight power of internal movement. Slight degrees of upward, downward, and external movements are possible in the right eye, and internal deviation is about equal to that of the left. Pupils react normally. She cannot close her jaws, nor can she push her lower jaw forward or to either side. Movements of face can be made at command. The tongue can be protruded through the lips but not many times in succession. Palate movements are weak. Head tends to fall forward on the chest. Arm and leg movements are good. Sterno-mastoids still show the myasthenic reaction. Knee- and wrist-jerks are brisk and equal. Plantars give a flexor response. There is no sphincter trouble.

June 29.—Dr. F. Buzzard saw the case with Dr. Head. There is no impairment of resonance over the upper part of the sternum. There are no glandular enlargements. Thyroid region appears normal. The temporal muscles are definitely wasted.

July 1.—Differential count of white blood corpuscles made by Dr. Grünbaum.

Six hundred cells classified. Neutrophile polymorphonuclear, 73 per cent.; lymphocytes, 19 per cent.; hyaline, 4·25 per cent.; eosinophile, 3·25 per cent.; basophile, ·5 per cent.

July 11.—Knee- and wrist-jerks cannot be exhausted by frequent taps. The patient's condition remained much the same although she lost weight and wasted considerably, until September 23, when she had some severe choking fits, became very dyspnœic, and died.

The autopsy was made twenty-four hours after death and *rigor mortis* was present. The body was somewhat emaciated. Neither the brain nor the spinal cord showed any change to the naked eye. The region of the thymus gland was occupied by fatty tissue. The thyroid weighed 20·5 grammes and was of natural consistence and appearance. Pancreas weighed 2 ozs. and seemed normal. Spleen was firm, and weighed 8½ ozs. Kidneys were healthy except for a small yellow nodule in one pyramid, the size of a pin's head. Adrenals presented a few small fibrous nodules but were otherwise normal. The liver weighed 2 lbs. 9 ozs. and seemed healthy. The heart was natural except for a small yellow atheromatous patch on the ventricular flap of the mitral valve. The lungs contained air throughout, except the base of the left lower lobe which was plum-coloured and sank in water. The right ovary was cystic. The muscles had a healthy appearance.

MICROSCOPICAL.

Central nervous system.—The Nissl and logwood-eosin methods were used for the examination of the motor cortex, the oculo-motor, facial and hypoglossal nuclei and the ganglion cells of the cord. The large majority of cells in all these regions were of a perfectly healthy type, and if a few were slightly swollen and pale there was certainly nothing like a true chromatolysis or atrophy to be seen. Weigert-Pal specimens of the motor cortex, the brain-stem and spinal cord were remarkable only for their normal appearance. Sections stained by Busch's method from the same regions must also be passed as normal, although a few isolated black dots were scattered about in the white matter. No lymphorrhages were seen in the brain or spinal cord.

Peripheral nerves.—Sections stained by the Weigert-Pal and Busch methods were made from the trigeminal, sciatic and ulnar nerves, but these presented no evidence of pathological change. Other sections stained by the logwood-eosin method showed no obvious abnormality.

External ocular muscles.—The appearances presented by sections of these muscles were practically identical with those found in Case 1. Lymphorrhages were unusually small but fairly numerous and were unassociated with any other changes either in the muscular, nervous, or connective tissue elements.

Skeletal muscles.—Portions of many of these muscles were examined, but only in a few were lymphorrhages observed. Excellent examples, however, were obtained in the rectus abdominis, and one of these in longitudinal section is represented in Plate II., fig. 1. This one measures about ·7 mm. in length and ·25 mm. in its greatest breadth. An empty capillary, represented by its endothelial lining, was seen to traverse the centre of the mass of lymphocytes and the neighbouring fibres appeared to be perfectly healthy.

Apart from the presence of lymphorrhages only a few sections showed any other pathological conditions and these were much the same as have been described in Cases 1 and 2, but perhaps in a few instances of greater intensity. Rounded, swollen fibres with hyaline and homogeneous sarcoplasm were present, but in some the change had gone a step further, and alterations in the refractive properties of the central portions of the fibre, with pericentral vacuolation, or central nucleation and vacuolation, were occasionally observed and reminded one of the earliest stages of certain forms of muscular atrophy. With these exceptions, and they were comparatively rare, the muscles presented a very healthy appearance.

Cardiac muscle.—Attention must be drawn to the condition seen in Plate II., fig. 2, which pictures a capillary vessel crowded with white blood corpuscles, chiefly of the lymphocytic variety and unaccompanied by any red corpuscles. Some of these cells had escaped into the neighbouring tissues, but the specimen is unique in my series, and important in that it suggests the condition which precedes the production of a lymphorrhage.

No changes were observed in the heart muscle fibres themselves.

Liver.—The changes found in this organ were rather difficult to describe. Scattered here and there in the midst of healthy hepatic tissue were small areas from which the liver cells had disappeared and were replaced by necrotic *débris*, together with a number of small round cells, some of which had the appearance of lymphocytes and others had irregularly-shaped nuclei.

Dilated spaces containing a few cellular elements were occasionally seen in the substance of Glisson's capsule. As far as size, distribution and general appearances are concerned they were compatible with what may be supposed to be the ultimate remains of lymphorrhages, and the presence of the small cells, some of which may also be undergoing degenerative changes, lends some support to this view. At the same time I think it wiser to abstain from expressing any decided view as to their origin.

The greater part of the liver tissue appeared to be healthy, both microscopically and macroscopically.

Adrenal bodies.—In the case of these organs there could be no doubt as to the presence of lymphorrhages, which were numerous and in some cases of considerable size. They occurred in most instances in the inner or central parts of the cortex, although the medullary region was not spared entirely. Beyond the mechanical destruction of the glandular cells in the immediate neighbourhood, the adrenal tissue did not appear to be affected by their presence and would pass muster as healthy.

The spleen, pancreas and kidneys.—These viscera presented no pathological changes on microscopial examination.

The thyroid gland.—Dr. Chalmers Watson who kindly examined the slides prepared from this gland reports that there were: (1) Distinct interstitial fibrosis; (2) numerous areas of cellular infiltration resembling small abscesses (lymphorrhages); (3) a large area of colloid degeneration of the fibrous stroma in one part; (4) proliferation of the epithelium with formation of new vesicles in some regions; and (5) enlargement of spaces as seen in simple goitre.

Thymus gland.—Dr. Dudgeon reports that only islets of gland tissues could be seen, scattered throughout dense connective and adipose tissue. In some places the adipose tissue was surrounded by glandular structure. The cortex could not be distinguished from the medulla and some endothelial proliferation was present. The concentric corpuscles were mostly in good condition, but some had undergone both granular and calcareous degeneration. No eosinophile cells were seen.

Case 4.—H. B., a schoolmaster, aged 40, was admitted to the National Hospital in October, 1899, under the care of Dr. T. Buzzard, and the following account is reproduced from the *British Medical Journal* of March 3, 1900, in which the clinical features of this case, along with another, were published.

Family history.—The patient's mother died of phthisis. His father, who is alive, aged 63, has suffered from muscular rheumatism. The grandparents on both sides are long-lived. The patient had four brothers and sisters, of whom a brother three years younger than himself is alive and strong; a sister died of scarlatina, two died in infancy. He has three children, aged 14, 10 and 8, all of whom are fairly strong, and there have been no deaths, no miscarriages. No similar condition to the patient's is known to exist among his relations. There is a rheumatic tendency in his father's family. A paternal uncle died in a lunatic asylum; a paternal aunt, aged 60, of "creeping paralysis."

Personal history. — Although never robust he was quite healthy till 1892. Twenty-eight years ago he had scarlatina, otherwise he has never had a day's illness until the present trouble. Syphilis can with certainty be excluded. There is no history of injury. He has not had a distinct attack of influenza. He is a total abstainer from alcohol, and not a heavy smoker. His work as a schoolmaster is hard and involves much speaking. His recreations have been chiefly of a sedentary nature, although he is fond of walking. He has had considerable worry during the last year or two.

Present illness.—Until 1892 he enjoyed good health. He then began to take a cold bath every morning, and is disposed to attribute his illness to this. About Christmas, 1892, after sawing wood, his little and ring fingers felt weak, but speedily recovered. The weakness recurred after playing the organ, and his right index finger would occasionally drop. In 1893 he occasionally saw double, and this trouble recurred in 1894. About this time his voice, after speaking a few minutes, would acquire a nasal character. His right eyelid, too, tended to droop. This weakness in fingers and eyelids occasionally recurred, but not in a very marked form, during the three following years. In the summer of 1897 he felt weakness in the lips and tongue, and spoke through his nose. After speaking some time the voice would become feeble. He again saw double. In the vacation he walked eight or ten miles a day without fatigue for several days. After this his right thigh became weak. He found that he had frequently to stop and rest it for a few minutes, after which he could go on walking. He had never noticed this difficulty before. In the winter of 1897-98 both arms and hands were easily fatigued, and the right leg felt weak. In July, 1898, his speech was again affected, especially in the evenings. He walked sixteen miles in one day during the vacation, and although he found difficulty in accomplishing the last few miles, this was due rather to trouble with his breathing than with his legs. At times he would suffer from palpitation, which was new to him. In October, 1898, whilst suffering from catarrh, he had difficulty in coughing and clearing his throat, and also in swallowing. This cold seems to have pulled him down very much, and he has never recovered the strength which he had before. In January, 1899, his right arm was so weak that he could not brush his hair or write upon the blackboard, but after two days' rest he was able to move the arm quite well. At this time there was dull, aching pain in the right arm and leg, especially after exertion, but also sometimes whilst lying still in bed. At Easter, 1899, he again had ptosis of the right lid, which has persisted in varying degree since. In June he gave up work. Diplopia and masticatory difficulty were then his chief troubles, but from time to time he would also suffer from dyspnœa, especially after exertion or emotion. From August he again improved, but was subject to recurrences of all his troubles, especially in very hot or cold weather. After a considerable rest his symptoms almost entirely disappeared.

Condition on admission (October, 1899).—His face is somewhat expressionless, and there is right-sided ptosis. Intelligence is not impaired. After speaking a little his speech becomes slower, laboured, and indistinct, and the voice is nasal in character and gradually fails. He describes the lips and tongue as feeling tired. It is noticeable that the chest expands less and less with each succeeding breath. The change in speech would appear to depend upon weakness of the respiratory muscles as well as those of articulation. Diplopia is only present on extreme lateral deviation to the right, and then only if the eyes are maintained for some considerable time in this position. The optic discs are normal. Hearing, taste, smell, and the viscera are unaffected. The ptosis is more marked in the evening, and is present to a slight extent also in the left eyelid; it is not always present. Two days after admission, when examined in the morning before rising, instead of ptosis there was actually retraction of the upper lids, so that a portion of the sclerotic was exposed above the cornea. Directly this condition was remarked upon the lid began to droop, illustrating what he says is one of the most characteristic features of his complaint, namely, that when his attention is drawn to any particular symptom, it will become exaggerated. He cannot voluntarily open his eyes widely, but when interested in conversation the lid moves upwards to a greater extent than he is capable of producing by any act of the will.

Ophthalmological examination.—Mr. Gunn, during his examination, remarked this incident, and attributed it to the action of Müller's muscle. Not only is he unable to open his eyes widely, but if he looks steadily upwards at an object held above his line of vision, the upper lids gradually droop lower and lower until the eyes are almost closed. Mr. Gunn reports: "Downward movement and convergence are good; upward movement defective; both external recti are weak. The pupils are equal, of moderate size, and react perfectly to light and on convergence; they dilate upon appealing to the patient's emotions, and occasionally upon mental concentration after prolonged convergence they show a great tendency to oscillatory movements."

Laryngological examination.—Sir Felix Semon, who was so good as to examine the palate and larynx, reports as follows: "The condition of the motor movements of the palate corresponds to what has been observed in the other voluntary muscles upon voluntary exertion. At first the palate is fairly well and symmetrically raised on phonating the letter A; gradually, however, as he repeats phonating, the movements become less and less marked, and finally hardly any raising of the palate is observed.

"On laryngoscopic examination, at first the larynx appears perfectly normal, except perhaps that on deep inspiration the vocal cords do not separate quite as much as in the ordinary condition; but as the patient, previous to the commencement of the examination, complained spontaneously of the shortness of breath, this might have been due to his general inability to take a long breath. Closure of the glottis on phonation was, and remained, perfect; on repeating his endeavours at breathing deeply, the glottis certainly finally opened less than at first. It is very probable, however, that this phenomenon was only part and parcel of the general exhaustion."

Muscular System.—He is quite unable to raise the eyebrows and wrinkle the forehead, and can only frown slightly. He can close his eyes, but cannot screw them up firmly. He can whistle and show his teeth, but movement of the upper lip is feeble. Contraction of masseters, temporals and pterygoids is feeble. He says that the jaw will frequently drop, the muscles apparently being unable to support it. The masticatory muscles become tired in the act of eating. The lips feel weak after talking, and at night saliva will dribble from his mouth. Occasionally he feels twitching sensations about the face, though no movements have been noticed, but he is awakened sometimes by a champing movement of the lower jaw. There is at present no difficulty in swallowing. The tongue is protruded straight, promptly, and to its full extent. It is symmetrical, and the movements are good, but easily exhausted.

The muscular power of this patient is subject to great diurnal variations. Towards evening he often sits with his hands clasped behind his neck supporting his head, as though the neck were weak. There is a tendency for the head to fall backwards. He cannot sit up for very long without feeling the back weak. He cannot sit up in bed without using his hands. He has some difficulty in turning in bed.

The rapidity with which the respiratory muscles are fatigued is demonstrated when he reads. Respiration is abdominal in type. During respiration there is almost no thoracic expansion, the difference in circumference of the chest between extreme voluntary inspiration and expiration amounting to less than half an inch. No weakness of the diaphragm can be detected.

The movements of the upper extremities are feeble, the right more so than the left; the dynamometer grasp, right 45, left 50 (average man 160). The ready way in which the muscles of the hand become fatigued is demonstrated by asking him to button and unbutton his waistcoat repeatedly. As regards his writing, he begins to write rapidly, forming his letters well. After a few lines the writing becomes slower and laboured, and the letters are not so well formed. Finally he comes to a stop, more or less suddenly, unable to write further. After a minute or two's rest he is able to write again as well as at first.

He can raise his arms above his head, but cannot maintain them in this position for more than half a minute. They gradually fall; he feels as if heavy weights were attached to them. Again, after a short rest he performs the movement quite well. If requested to lift his arms quickly above his head several times in succession he will do this, but at each effort the arms are lifted less high, and after doing this seven or eight times they cannot be lifted at all, but hang by his sides. After a rest of from thirty to sixty seconds they can be lifted nearly as high as they were at first. All movements of the lower extremities are well and powerfully performed, but the right leg especially is easily fatigued. There is no muscular atrophy, nor are there any fibrillary twitchings. The muscles respond fairly well to direct stimulation with a percussion hammer and no diminution in the activity of the response results from repeated blows.

Sensation.—A dull aching in the neck, back, arms, and right leg, occurring particularly after exertion, but also when lying in bed, is sometimes present. No disturbances of cutaneous sensibility can be ascertained.

Reflexes.—The arm-jerks are present, feeble. The knee-jerks are present and equal, usually a little more active than the average. The patella-jerk is not depressed. Repeated attempts were made to determine whether the knee-jerk diminished after frequent tapping. On one occasion the tendon was tapped continuously for a quarter of an hour, but at the end of that time quite as good a knee-jerk was obtained as at first. No ankle clonus is present. No abdominal reflex was obtained. The plantar reflex is present, with sluggish flexor response.

Sphincters.—The bowels are regular. There is no trouble in commencing micturition, and the stream is well maintained.

Cranium and spinal column.—There is no abnormality. The gait is natural. After walking several times up and down the ward nothing abnormal is noticed. No vasomotor or trophic changes have been discovered.

Of great importance and interest are the results obtained by electrical examination. The muscles respond to faradism in varying degree on different occasions. For instance, if an electrode be placed upon the motor point of the biceps a prompt and good contraction is obtained, except when the muscle has been fatigued by voluntary exertion. The initial contraction in certain muscles is very much poorer than in others, and some muscles tire very much more easily than others. As the result of faradic stimulation, voluntary power of contracting the muscle stimulated is much diminished and a feeling of fatigue induced. As regards galvanism, after repeated stimulation the muscular contractions become less, but the muscle cannot be tired to the same extent as is the case with the faradic current. No polar change can be demonstrated.

This patient left the hospital in the early part of 1900, but remained more or less in the same condition for the next few years. During the year 1903 he was hardly able to walk from one room to another, and always went out in a bath-chair. Every few weeks he would suffer from severe paroxysms of dyspnœa, and he ultimately died of respiratory failure on January 24, 1904. Owing to the kindness of Dr. Morrow, of Enfield, I was able to perform an autopsy twenty-two hours after death at the patient's house.

The body was extremely emaciated, but the muscles, though poor in size, preserved their natural contour and presented no evidence of local atrophy.

The brain and spinal cord with their meninges and vessels appeared normal to the naked eye. Portions of only a few of the larger muscles and nerves were removed for the examination, and unfortunately the ocular muscles were not among this number.

The heart, lungs, spleen, liver and kidneys showed no signs of disease. The pancreas had undergone *post-mortem* digestion.

The stomach and intestines were normal, and one or two spleniculi were found attached to the mesentery.

The thyroid was natural in size and appearance.

The thymus gland was rather unusually well developed, weighing 9·5 grammes after the adjacent fatty tissue had been stripped off.

MICROSCOPICAL NERVOUS SYSTEM.

The cells of the motor cortex were in excellent condition, and the same remark applies to those of the bulbar nuclei. A few small recent hæmorrhages were seen in the floor of the fourth ventricle. A careful examination of the cells of the cord was made, and it was thought that they appeared smaller than the normal on the average, but the difference was slight if present, and in other respects they lacked none of the healthy characteristics. A few capillary hæmorrhages were noted in the gray matter of the anterior horns, but these were quite recent and associated with considerable congestion of the vessels.

The Weigert-Pal and Busch methods showed no alteration in myelinated fibres either in the central or peripheral nervous system, and sections of the cord and sciatic and ulnar nerves stained by logwood and eosin showed no pathological conditions.

Muscular system.—Owing to the circumstances under which the *post-mortem* examination was made only a few small pieces of muscle were investigated microscopically, and these came from the erectores spinæ, the left pronator radii teres and the left biceps femoris. In the sections examined, only one or two small lymphorrhages were found and the fibres as a whole were of healthy size and appearance. A certain number, however, were present in which a rounded swollen contour and homogeneous sarcoplasm were noted. No changes were detected in the vessels, connective tissue, or muscle spindles.

Spleen.—There was no thickening of sheath or septa and the relation of the pulp to the fibrous constituents was about normal. Malpighian bodies plentiful and well marked. A spleniculus was also cut and the sections presented similar appearances.

Pancreas.—This organ had undergone so much digestion that it was of no use from an histological point of view. In the loose areolar tissue surrounding the pancreas masses of lymphoid tissue were seen under the microscope having an appearance similar to the islets of thymus remains, but containing no Hassall's bodies. Some of the vessels in this tissue were closely surrounded by clusters of lymphocytes, and some had their lumina blocked with the same kind of cells.

Adrenals.—Numerous small lymphorrhages were seen in the sections of otherwise healthy glands. One of these is depicted in Plate I., fig. 2.

Thymus.—Report by Dr. Dudgeon. The gland is large and the cellular constituents are in good condition. The cortex and medulla can be differentiated from each other, although the distinction is not so well marked as in the case of the normal organ. There is some increase in the fibrous stroma, and adipose tissue has replaced the glandular tissue in several parts. Many of the concentric corpuscles are very large and calcified, while others give evidence of granular degeneration. No eosinophiles are to be seen.

Case 5.—A man, aged 40, was admitted to the London Hospital, under the care of Dr. C. Wall, on February 10, 1905, with the history of a month's illness.

He was a foreman for a mineral water company. His wife was healthy and had had three children and two miscarriages.

He had suffered from "malaria" fourteen years previously, and since that time was subject to incontinence of fæces after taking aperient medicine.

Present illness.—One month ago he noticed such weakness of his neck that he could not hold his head up straight. The eyelids tended to droop, and he was unable to lift his arms above his head. Difficulty with articulation, mastication and deglutition was also observed, as well as considerable trouble in keeping food from collecting between his teeth and cheeks. Articulation would become worse after he had talked for a while, and diplopia was present at times.

Condition on admission.—The heart, lungs, and abdominal viscera appeared healthy on examination.

The optic discs were healthy. There was partial ptosis of both upper eyelids, weakness of the left external rectus and right internal rectus muscles. There was no nystagmus.

He was unable to protrude the tongue beyond the teeth, but no tremor was observed. All the muscles of the neck were paretic and he was incapable of raising his arms above his head.

Romberg's sign was absent. The knee-jerks were present and equal, and the plantars flexor in type.

February 22, 1905.—Patient had an attack of dyspnœa last night, and a bad fit of choking to-day while eating pudding. He can talk but little without becoming exhausted.

Electrical reactions. The myasthenic reaction is present in the trapezius of both sides, in the pectoralis major, the biceps and flexors of wrist on the right side. The triceps and deltoid muscles did not give the reaction, but it was present in the facial muscles, especially on the left side.

February 23.—Diplopia is marked, but the pupils react normally. The left upper eyelid droops more than the right.

The movements of the right eye are good, but there is complete paralysis of the left external rectus.

February 25.—Swallowing is best in the early morning, and becomes more difficult as the day goes on. He has had two more bad choking fits.

February 27.—There is marked improvement in the ptosis, swallowing and dyspnœa, and the double vision is less than it has been for weeks.

The patient's condition varied from day to day until April 6, when he had increasing difficulty in breathing, and was unable to cough up the mucus from his bronchial tubes. On the 7th his respiratory muscles became progressively weaker, and death took place from respiratory failure.

The autopsy I was kindly allowed to make at the London Hospital fifteen hours after death. The body had been frozen and *rigor mortis* was marked. No evidence of local atrophy or general emaciation was found on inspection.

The brain and spinal cord with their coverings appeared healthy to the naked eye. The pituitary body was normal. Portions of several peripheral nerves and of many muscles were kept for microscopical examination, but to the naked eye presented a natural appearance.

Thorax.—Behind the manubrium lay a large mass which on removal weighed 59·4 grammes. The upper part of this mass was firm and adherent to the subjacent vessels, the lower part was not so adherent and was softer and cystic. On cutting into the upper part it was found to be soft and creamy in the centre, and slide smears were prepared of the pus-like material. Closer examination of the lower portion showed that it was a thin-walled multilocular cyst containing a slightly cloudy fluid.

The lungs were healthy except for an old scar at the left apex.

The heart weighed 10 ounces and was free from any evidence of disease.

There were no enlarged bronchial or tracheal glands. The thyroid weighed 13 grammes and was pale in colour, but otherwise natural.

Abdomen.—The liver looked healthy and weighed 3½ lbs.

The spleen weighed 9½ ounces, was firm and showed prominent malpighian bodies.

The kidneys, pancreas, adrenals, stomach, and intestines, were all examined with negative results. The mesenteric glands and Peyer's patches were prominent without being unduly enlarged. A portion of the left femur was removed with the intention of examining the marrow, but it may be stated at once that this proved of negative value owing to the fatty, non-cellular condition of the latter.

MICROSCOPICAL.

Owing to the acute course of the disease in this patient—the whole illness only lasted about three months—it was thought that the examination of the cells of the central nervous system would be of particular interest, and a number of sections, stained by Nissl's method, were prepared from the motor cortex, the oculomotor and bulbar regions, and from various levels of the spinal cord. After a careful investigation of these it was impossible to state that any definite pathological changes were present, although a certain number of anterior horn cells were somewhat deficient in tigroid. On the other hand, the oculomotor nuclei would pass as typical examples of healthy cells possessing abundant tigroid and central nucleation.

Serial sections of a cervical posterior root ganglion were stained by the same method and presented an entirely normal appearance.

As in the preceding cases neither the Weigert-Pal nor the Busch methods were successful in revealing any morbid condition in the central nervous system or in the peripheral nerves. Several of the latter were teased, but no abnormal fibres were seen.

Muscular system.—The ocular muscles contained several small and one or two larger lymphorrhages which differed in no particular from those seen in other cases.

The skeletal muscles, from a number of which sections were cut and stained, presented some features peculiar to this case and deserving of special mention.

Lymphorrhages of the usual type and of varying size were found in a small proportion of the sections examined and were not always confined to the muscle fibres, as in one or two instances they occurred in the fibrous sheath of the muscle. In addition to the ordinary lymphorrhages a condition was found in a number of serial sections taken from a portion of the triceps muscle which is represented in Plate I., fig. 3. A cellular condition is associated in this instance with marked changes, in the form of swelling and protoplasmic disintegration, in the neighbouring muscle fibres. This reaction to the cellular exudation is unique among my specimens and is rendered more interesting by the fact that an examination of the cellular elements of the exudation showed that they differed from the cells usually seen in lymphorrhages. Some of the cells were probably true polymorphonuclear leucocytes but the nature of many of the others could not be definitely ascertained. Their size was very much that of lymphocytes, although some were larger, but they presented more clear protoplasm and contained nuclei which in many instances appeared to be undergoing division into two or four parts. Owing to the fact that they lay thickly together in celloidin sections accurate examination of their characters was impossible, and these facts can only be recorded without any attempt being made to define their true significance. It will be noticed that some of the altered muscle fibres were actually invaded by the cells, a condition which does not apparently obtain in the ordinary type of lymphorrhage.

In the neighbourhood of a small lymphorrhage, in another section, some of the fibres presented an appearance which is occasionally seen in conditions of very early muscular atrophy of toxic origin. In transverse section the fibre is rounded and in the centre is a dark core surrounded by a concentric ring of lighter hue; as far as can be ascertained the appearance is produced by an alteration in the refractive properties of the central and pericentric portions of the plasma, and not to the presence of nuclear elements on the one hand and of disintegration on the other.

In longitudinal sections evidence of early atrophy was also forthcoming here and there in the form of localised swelling in a fibre and marked proliferation of the sarcolemma nuclei.

While attention is necessarily drawn to the morbid conditions discovered, it must be clearly understood that in this case, as in the others of my series, the large majority of fibres could not in any way be differentiated from those of healthy muscle.

Liver.—In otherwise normal sections a few well-marked lymphorrhages were seen, generally situated round and about the biliary ducts or blood-vessels.

Adrenals.—Microscopical preparations of these organs contained only one or two doubtful lymphorrhages of minute size, and the only feature of note was the intense congestion of all the blood-vessels associated with pallor of the gland cell nuclei.

Pancreas.—The histological appearances of this organ were of a healthy character.

Thyroid.—No lymphorrhages were found and no other abnormal features noted.

Thymus.—The examination of this organ included the preparation of: (1) smears from the semi-fluid matter contained in the centre of the upper solid portion, and from the liquid contained in the cystic portion of the gland; and (2) sections of the upper portion and of the multilocular cysts. These were stained by logwood and eosin, by Jenner's stain and by Leishman's stain.

The more solid part of the organ consisted of closely packed masses of ordinary thymus gland cells separated by dense bands of connective tissue; it contained no traces of Hassall's bodies. In the smears taken from this part the prevailing cell was of the lymphatic type, but there were also a considerable number of eosinophile cells of large size. The cystic portion resembled the solid part except for the fact that the dense masses of cells were replaced by a thin fluid containing similar cells in much smaller numbers. Owing to the pressure of this fluid the cyst walls appeared to have a definite lining membrane, but this was not of true cellular structure. In the midst of the fibrous strands composing the cyst walls many lymphocytic cells were entangled, sometimes in large clumps, and a fair number of eosinophile cells were also observed. One or two doubtful remnants of concentric corpuscles were noted.

Pituitary body.—Sections showed no abnormality beyond some slight colloid degeneration in the glandular part of the body.

CLINICAL SUMMARY.

The five cases here recorded, although varying very considerably in their duration, all presented the typical motor phenomena which we associate with myasthenia gravis, and each one exhibited the bulbar symptoms which at one time gave the name "asthenic bulbar palsy" to the disease. The disorders of motion are now so well known that it is unnecessary to dwell upon them here, and attention will be at once directed to certain other disorders presented by some of my cases which are more rare and less well recognised.

SENSORY PHENOMENA.

In that excellent monograph on myasthenia published by Campbell and Bramwell (3) in 1900, the sensory side of the disease is dismissed in the few words "there are no sensory symptoms." In the five years which have elapsed since those words were written, a large number of cases of myasthenia have been recognised, carefully investigated and recorded, with the result that that statement no longer holds good. Subjective pains and paræsthesiæ of various kinds have been mentioned in connection with a number of cases, but as far as I am aware no case of the disease has presented such startling disorders of cutaneous sensibility as were found in Case 1 of this series. A reference to the charts will show that the relative analgesia and anæsthesia in this patient was progressively distributed over those areas of skin which nearly always show the same changes in cases of tabes dorsalis. When the reader remembers that these changes were associated in this patient with "lightning" pains, with absent arm-jerks, and with diminished knee-jerks, he will not be surprised to learn that the possibility and even the probability of myasthenia being combined with early tabes was entertained during life.

The negative results obtained in a careful search for microscopical evidence of the latter disease, allow us to draw one of two alternative conclusions. The sensory disturbances are occasional, if rare, symptoms of the morbid process which we designate by the name "myasthenia," or the patient had a "functional tabes," by which is meant the disease in that early stage where the disorder of function precedes the anatomical lesion, if such a stage actually exists.

The second of these two alternative propositions is one which it is impossible to support with any material evidence at the present time, but the first may be considered in the light of contrast with other cases. Case 2 complained of intermittent sharp pains similar in character to those described by Case 1, and had in addition a small patch of relative analgesia and anæsthesia during a period of the time she was under observation. Senator (7) describes a case in which there was anæsthesia of the chin, and Burr (2) has mentioned the presence of some very interesting visual disturbances in one of the cases which he has reported. It can hardly be denied, therefore, that sensory phenomena may take a prominent place in the symptomatology of the disease, and their presence can no longer be held to be sufficient to throw doubt upon the diagnosis.

The explanation of their occurrence is a most difficult problem, but hardly more so than that of the motor phenomena. Some reference to this subject will be made in discussing the question of pathogenesis.

In this connection it may be interesting to mention that an undoubted case of myasthenia which was under my observation for two or three years, presented unmistakable evidence of mental changes which varied in intensity from time to time. Melancholia with delusions of persecution and some suicidal tendencies were very marked for short periods, and I have in my possession letters from the patient which illustrate well the morbid condition of his mind. Unfortunately I was unable to obtain an autopsy at his death. This experience again makes one hesitate to accept the dictum of Hun (4) that "mental abnormalities play no part in this disease."

MUSCULAR ATROPHY.

The occurrence of localised muscular wasting in myasthenia has been doubted, but recent records favour the view that it is certainly present in some cases. The tongue is a favourite site for atrophic change, and in Dr. Head's case there was definite wasting of the temporal muscles. Although I have never had the opportunity of examining histologically any markedly wasted muscle in this disease, I am satisfied from clinical observations that such wasting does occur in some instances.

MORBID ANATOMY.

In studying the records of cases of myasthenia which have been examined after death, it is found that in a large number the results have been negative, or variable and trifling, but that in a smaller number, and these belong to recent years, attention has been drawn to the existence of changes in the thymus gland and to the presence of lymphocytic deposits in the muscles. A considerable variety of thymus changes have been described, including so-called thymus tumours (possibly lymphosarcomata) and different degrees of thymus enlargement, and the view has been held that the lymphoid tissue found in the muscles has been of the nature of metastases. My cases show that abnormalities of the thymus are *not constant* features in this disease, and therefore that the lymphocytic deposits, which occur independently of them, are probably not in any way a secondary condition. On the other hand my experience affords reasonable grounds for believing that lymphorrhages are constantly present in the muscles or other organs in cases of myasthenia. That they existed in all five of my cases was only established by a diligent search through some thousands of sections, and for that reason it would be unwise to assume that they were absent from the cases of other observers who had not been led to conduct a similar careful investigation with the same object. Moreover, it is interesting to note that since their presence was first described their occurrence has been reported in an increasing number of cases. I would suggest that this number will be still greater when it is realised that a persistent thymus or a mediastinal tumour is not a necessary concomitant of the lymphorrhages. An exhaustive examination of all the muscles is too laborious a task for any investigator to undertake, but it is fairly easy thoroughly to search the ocular muscles and a number of selected samples from various skeletal muscles. The presence of lymphorrhages in other organs, described here for the first time, renders the undertaking still less formidable.

Although the occurrence of a thymus gland of unusual size or with pathological features is not, as we have seen, a constant factor, yet its frequency is so considerable that it will be impossible to disregard it in discussing the pathogenesis of the disease.

Out of my five cases one presented a very large thymus gland which had undergone changes partly proliferative and partly degenerative in nature, another exhibited considerable simple hypertrophy of the gland, a third a minor degree of the same change, while in the two remaining patients were found only those islets of lymphoid tissue which are commonly present in the adult human being.

Lymphorrhages were present in varying numbers in all five cases, but were by no means confined to the muscles. In several cases they were seen in the heart, liver, adrenals and thyroid gland. A considerable increase of round cells in parts of the kidney of at least one case was evident, but it was difficult to say whether the condition was identical with the lymphorrhages of other organs. In one instance (Case 1) there were scattered small foci of lymphocytes around vessels in the substance of several posterior root ganglia, and these I believe, after some hesitation, to be small lymphorrhages.

What is the origin and what is the fate of a lymphorrhage? To these questions I find it impossible to give a definite answer. The impressions gained from examining a large number of these cell deposits inclines one to the view that they arise in some way from the capillary blood-vessels, and this feeling is strengthened by the fact that no evidence of any lymphatic disease has been forthcoming. At the same time we are shaken in our belief by the absence of blood changes during life and by the knowledge that the lymphocytes are not the most migratory of blood cells.

Certain appearances which would seem to give some support to the view that lymphorrhages are of vascular origin have been described in the preceding records of the cases but may be referred to again in this place.

In the first place the close association of the lymphocytic exudation with a small blood-vessel has been noted repeatedly. In Plate II., fig. 2, a capillary vessel is seen to be engorged at one point with white corpuscles, some of which have escaped into the surrounding tissues. In Case 2 attention has been directed to a cardiac blood-vessel full of red corpuscles, but surrounded by a mixed serous and lymphocytic exudation. A somewhat similar condition was seen in the liver sections of Case 3, in which dilated spaces containing amorphous *débris* and a few white cells were found in parts of Glisson's capsule. It would be premature to argue dogmatically from those preparations, but they certainly seem to throw some light upon the formation of these cellular deposits.

With regard to the fate of the lymphorrhages we can only suppose that they are transitory phenomena, since they are not more numerous, if as numerous, in long-standing cases as in those which run an acute and rapidly fatal course. Does the prevalence of lymphorrhages in a muscle bear any relation to the functional activity of that muscle? The data at my disposal are inadequate for the purpose of forming any opinion on this question.

The possibility that lymphorrhages might occur in other conditions was one which naturally arose, and an effort was made to settle the point by examining sections of muscles obtained from various other diseases of muscular or nervous origin. The ocular muscles from a case of pontine tumour, in which there had been ophthalmoplegia for many months before death, were investigated with negative results. In a variety of other conditions the skeletal muscles were searched, but in only one instance was there found any appearances resembling the lymphorrhages described above. The case was one of amyotrophic lateral sclerosis which ran a very atypical clinical course, and in which the lymphocytic deposits were associated with extensive and unusual changes in the muscular fibres. In another example of the same disease no lymphorrhages were observed.

Although the lymphorrhages appear to be the most striking of the anatomical changes in myasthenia, yet it is desirable to point out that in every one of my cases some of the muscles have shown abnormal appearances in a small proportion of their fibres. These abnormalities have not always been identical, but they have all been of the character which one associates with an early stage of muscular atrophy, and suggest that they would have resulted in grave muscular degeneration had the morbid process at work been further encouraged. They certainly make the occasional occurrence of true muscular atrophy in this disease by no means surprising.

Attention having been directed to certain positive factors in the morbid anatomy it will be well to emphasise the untrustworthiness of certain negative factors. For instance, there can be little doubt that the methods at our disposal for the examination of the axis cylinders in nerve trunks, and more especially the terminations of neurofibrils in their muscular connections, are inadequate, and it is impossible, therefore, to feel certain that these important structures do not show definite, but hitherto undetected, alterations. At the same time we should expect that permanent structural changes in these tissues would lead to some recognisable reactions, either in the myelin sheaths, or in the neurone cells, and of such there is no evidence.

The discovery of small lymphoid masses resembling thymic islets, except for the absence of Hassall's corpuscles, in the peripancreatic connective tissue of Case 4, makes one regret that more of this tissue in various parts of the body was not examined.

PATHOGENESIS.

Great as has been the increase in our knowledge of myasthenia within recent years, it is undeniable that the pathogenesis of the disease is still far from clear, and it is not my intention to discuss the problems with which we are faced at any length. At the same time it may not be unprofitable to refer to some points which are suggested by the cases just described, as well as to some further points observed in other cases, and to review them in the light of recent theories on muscular and glandular physiology.

A few years ago, Dr. H. D. Singer and I carried out some investigations on two cases of myasthenia in the National Hospital, some results of which I will briefly summarise here.

(*a*) Biceps humeri. With faradic current, the muscle rapidly ceased to be tetanised, the subsequent contractions being intermittent for a time and then ceasing altogether. By this time voluntary power was much diminished but not lost, the biceps-jerk remained unchanged, the galvanic reaction was brisk, and direct excitability to percussion was as lively as before.

(*b*) Same muscle. The muscle was rapidly tired out by voluntary action. Diminished faradic excitability was then present. The galvanic reaction, the tendon-jerk and direct excitability were unaltered.

(*c*) Same muscle. Rapidly repeated galvanic shocks for two minutes produced no diminution in the briskness or size of the response.

(*d*) Same muscle. Faradism with moderate strength of current was continued until no response was obtained. An increase in the strength of current immediately produced a contraction which was rapidly exhausted.

(*e*) Same muscle. The tendon-jerk was not diminished by rapid repetition for a considerable time.

(*f*) Attempts were made to ascertain the effect of exhaustion of one group of muscles upon the activity of another but the results were variable. General clinical observations seem to show, however, that there is a general reduction of power produced by local exhaustion.

(*g*) The same muscle in another patient, very weak and quickly tired. Tendon-jerk absent. Direct excitability to percussion less than normal.

After exhaustion by faradism there was still further loss of voluntary power. After further exhaustion by voluntary action there was dimished reaction to faradism. Galvanic response good and brisk.

The muscle was exhausted by faradism and then by voluntary action. When it ceased to respond to volition it had regained some response to faradism; galvanic response still good.

The above observations, which were made at a number of different sittings, admit of course, of various interpretations, but they appear to me to suggest certain conclusions. In the first place the exhaustion of a myasthenic muscle is always a relative phenomenon, and bears a definite relation to the nature and probably to the strength of stimulus employed. In the second place the liability to exhaustion is much more pronounced in the case of tetanising stimuli, such as are employed in voluntary action and faradism, than in the case of stimuli producing single sharp contractions such as direct percussion, indirect excitation through the tendon and galvanism.

Thirdly, I would emphasise most strongly the fact that no comparison should be drawn between the phenomena observed in myasthenic muscles and those which we associate with the reaction of degeneration. In the former case a muscle which has lost its response to faradism gives the normal brisk response to the make and break of a galvanic current. In the latter case a muscle, irresponsive to induction currents, gives a slow worm-like contraction on applying or discontinuing the galvanic current.

Jotezko (5) has shown that the slow contraction just mentioned is an idiomuscular phenomenon, and this fact, taken in conjunction with those just mentioned, distinctly contravenes the theory that in myasthenia we have to deal with an exhaustion or liability to exhaustion on the part of the muscular nerve endings.

Botazzi (1) has advanced the view, which is supported by

Jotezko (6), that in muscle there are two contractile substances, a fibrillar and a protoplasmic. The fibrillar contraction is the short sharp response, such as is produced by the make or break of a galvanic current; the protoplasmic contraction is slow and resembles that which is seen in the reaction of degeneration. They can both be produced by the galvanic current, but the protoplasmic reaction requires a stronger stimulus than the fibrillar. After nerve section the muscle tends to return to an embryonic condition, in which the fibrillar substance diminishes and the sarcoplasm relatively increases. Assuming these views to be correct they would help to explain the paradoxical condition observed in the myasthenic reaction, and we might suggest that in the disease under consideration, some agent is at work which reduces the excitability of the protoplasmic more than that of the fibrillar constituents of the muscle.

Going a step further it may be asked, what evidence is there of the presence of a toxic agent of either exogenous or endogenous origin? So far as our knowledge goes there is no good reason for believing that any microbe is the causative agent of the disease; on the other hand the clinical features of myasthenia present many resemblances to those of what are called the internal secretion diseases and especially to Graves' disease and to Addison's disease. With that idea in my mind, the ductless and other glands have been the object of much attention in the histological examination of my cases, but the results have not been illuminating. It is true that several of the glands have been shown to be the site of lymphorrhages but these are so widespread in their occurrence in these cases that their presence can only be regarded as part of a general condition and not as the specific cause of morbid glandular function.

The frequent abnormalities in the thymus are interesting in this connection but their inconstancy is a bar to attaching too much importance to them. At the same time it is well to remember that experimental observations have led to the belief that the thymus has some excito-motor influence when injected into animals.

The fact that the thymus is mainly a lymphatic structure and that lymphorrhages form a prominent feature of the morbid anatomy of the disease, will doubtless lead to further investigation of the lymphatic system in myasthenia, and in this direction lies our best hope of solving the problem of its pathogenesis.

I have stated above that I was unable to estimate the relation of lymphorrhages to the functional activity of a muscle, but on clinical and anatomical grounds it would seem quite likely that some such relation may exist. The probability that lymphorrhages are temporary phenomena and the fact that muscles are irregularly, variably and sometimes suddenly affected in the course of the disease, suggest that in addition to a general morbid process there may also be local factors influencing its incidence. While admitting this I do not feel inclined to support the view that lymphorrhages exert a deleterious influence on muscular activity by the mechanical effect of their presence or by blocking the local lymph flow. As I have said in a former paper I prefer to regard them as the outward and visible sign of some more subtle—metabolic or chemical—influence which is exerting itself upon the function and in extreme and rare instances, upon the actual structure of muscular and other tissues.

It is necessary to assume that the toxic agent is capable of exerting an influence on the function of other tissues besides muscle in order to account for the sensory, mental, vasomotor and secretory disorders occasionally met with in the disease, but it is impossible to say at the present time whether a disorder of any one of these functions is always associated with the anatomical presence of lymphorrhages in the tissues subserving that function. In this connection I can only point with diffidence to the occurrence of cellular exudations, resembling in their histological features lymphorrhages of other tissues, in the posterior root ganglia of Case 1, the only patient of my series in whom diminution of cutaneous sensibility of root distribution was observed during life.

As a result of my experience and in order to remedy the defects in my own investigation I would advocate more attention being directed to the condition of the blood, the marrow, the lymphatic system, and last, but not least, to the fatty and connective tissues of the body in cases of this disease which come to *post-mortem* examination.

The conclusions to be drawn from this investigation may be briefly summarised thus :—

Clinical.

(1) That myasthenia gravis is a disease in which the symptoms are not always confined to the motor system, but may include others of sensory, mental or other origin.

Anatomical.

(2) That in all probability it has a definite and constant morbid anatomy constituted by the presence of widely distributed cellular, and sometimes serous, exudations (lymphorrhages) in the tissues and organs of the body.

(3) That slight muscle-fibre changes are frequent and severe muscular atrophy rare occurrences in the disease.

(4) That proliferative and degenerative changes in the thymus gland are frequently but not constantly met with.

Theoretical.

(5) That the symptoms of the disease are best explained by assuming the presence of some toxic, possibly autotoxic, agent which has a special influence on the protoplasmic constituent of voluntary muscle and a less specialised influence on the function of other tissues.

(6) That the relation of this toxin to the incidence of lymphorrhages and to thymic alterations is not clear.

REFERENCES.

(1) BOTAZZI. *Archiv. of Anat. u. Physiolog.* 1901, S. 377.
(2) BURR. *Journal of Nerv. and Mental Disease*, 1904, p. 28.
(3) CAMPBELL & BRAMWELL. BRAIN, 1900, p. 277.
(4) HUN. *Albany Medical Annals*, Jan., 1904, p. 28.
(5) JOTEZKO. *Compt. Rend. Soc. de Biologie*, 1899, p. 384.
(6) ,, *Journal de Neurolog.*, 1904, p. 221.
(7) SENATOR. *Neurolog. Centralblatt*, 1892, S. 168.

ATAXIA IN CHILDHOOD.

BY FREDERICK E. BATTEN, M.D.

Physician to the Hospital for Sick Children, Great Ormond Street, and Assistant Physician to the National Hospital.

INTRODUCTION.

CERTAIN well-known forms of ataxia occurring in children are commonly recognised, such as the ataxia of Friedreich's disease, the ataxia which occurs in tumours and lesions of the cerebellum and mid-brain, and in certain cases of diphtheritic paralysis. It is not with these well-known forms of ataxia that the present paper deals, but with certain cases of ataxia occurring in children which cannot be placed into one of the above groups. The instances on which the present paper is based have been seen, for the most part, at the Children's Hospital, Great Ormond Street, during the past ten years, and my thanks are due to several of my colleagues for not only allowing me to see patients under their care, but also for permission to refer to the same.

These cases, which may be classified as cerebellar diplegia, divide themselves into three groups.

(1) Cases in which ataxia has been noted early in life, and in which there is a tendency to gradual improvement (congenital cerebellar ataxia)

(2) Cases in which ataxia has suddenly developed after some acute illness in a child who formerly had been quite healthy (acute ataxia, encephalitis cerebelli).

(3) Cases in which a child has been healthy till a certain age, and then has gradually developed ataxia; cases in some points resembling Friedreich's disease, but differing from the classical picture of that disease (progressive cerebellar ataxia).

GROUP I.—CONGENITAL CEREBELLAR ATAXIA.

The following may be taken as the leading features of the disease :—

A child, whose backwardness in development is evidenced by the late period at which it learns to sit up, to walk, and to talk, is noticed when a few months old to "tremble" when using its hands ; to this little or no attention is usually paid, but when on sitting up it is noticed that the child shakes its head and the whole trunk is in constant oscillation, some defect is first recognised by the mother and advice is sought. Nystagmus of the eyes on attempting to fix an object may be present. When the child learns to stand, it is noticed that there is great unsteadiness of the legs, and on attempting to walk the legs are thrown about in a most ataxic manner. As the child learns to talk the words are pronounced in a slow, jerky, and monotonous manner. Many of these children are slow eaters, and, though they will swallow liquids well, swallow solids very slowly. They are clean in their habits, take an intelligent interest in all their surroundings, and are usually well nourished, and have good muscular power. As they grow, the ataxia of the arms and legs becomes less, their articulation improves, and in the slightest cases normal development takes place. The following cases may be taken as a fairly typical example of this group :—

Case 1.—D. S., aged 4, was first seen in February, 1899. She was the first and only child, and there had been no miscarriages. The labour had been prolonged, and instruments had been used to deliver the child. The mother was in good health during pregnancy. The child was brought to the hospital because she could not walk. She had not learnt to use her hands till she was 12 months old, and, though she could talk, her articulation was so defective that it was difficult to understand her. She had always had difficulty in swallowing, and had to be fed very slowly ; the difficulty was greater with solids than with liquids. She was intelligent and clean in her habits, and had never had any illness.

On examination the child was well formed and nourished ; she could stand and support her weight on the legs, but was unable to walk, and always tended to fall backwards. When an attempt was made to walk, the legs were thrown about in a wildly ataxic manner. The head and trunk were in constant movement, and, on attempting to grasp an object with the hands, the most marked inco-ordination and intention tremor were exhibited. The head was well formed, measuring 19½ inches in circumference, and there was no deformity of the skull. The movements of the eyes were good in all directions, and there was no nystagmus. The optic discs were normal. The hearing was good, the tongue protruded normally, and the hard and soft palate were normal. The back was straight, and there was no bony deformity either of the limbs, feet, or hands. The power of movement of the limbs was good, and there was no rigidity. The knee-jerks were brisk and equal. There was no ankle clonus, and the plantar reflexes usually gave extension of the great toe.

In October, 1899, she was seen again, and on this occasion could walk with great difficulty, and showed the same tendency to fall backwards. In February, 1900, her walking had improved, but she still tended to fall backwards. The head measured 20 ins. in circumference. The knee-jerks were brisk, and the plantar reflexes were noted as giving an indefinite extensor response, *i.e.*, the big toe extended, the small toes flexed.

In May, 1900, her walking had greatly improved. She could walk by herself, but still tended to fall backwards. She had much less inco-ordination of the hands, but the articulation was still very defective.

In March, 1901, she walked fairly well, with the trunk held forward in a position resembling that of paralysis agitans. The inco-ordination of the limbs and trunk was still present, but was comparatively slight. The knee-jerks remained active, and the plantar reflexes gave varying responses, sometimes extensor, sometimes flexor. The child was six years old when last seen, and appeared to have good intellectual development.

Case 2.—H. T., aged 3 years and 3 months when first seen in June, 1902. He had learnt to talk when 2 years old, and was bright and intelligent, but he had never learnt to walk.

He was unsteady with his hands and could only stand when his back was against the wall. He took food well and swallowed naturally. He was clean in his habits, but sometimes had an "accident," which greatly annoyed him. He was not passionate. He was the first of two children, and there had been no miscarriages. The mother stated that she was not in good health during pregnancy, but the labour was natural and rapid.

The child was a well-nourished, intelligent little boy, with the slow, monotonous articulation characteristic of this affection; but he had in addition a lisp, so that he omitted the first consonant of a word: for example, he said "oor" for door.

He stood unsteadily, and seemed always likely to fall. He walked with the knees flexed and close together, like a patient with cerebral diplegia of a mild type, but at the same time there was marked inco-ordination. The legs could not be said to be rigid, although the mother stated that they were so at times.

The knee-jerks were present, not very active; there was no ankle clonus, and the plantars gave a flexor response, though it was a response which was neither easy to obtain, nor definite in character. All the movements of the face were good, the movements of the eyes were good, the pupils were equal and reacted well to light; there was no nystagmus. The child sat up fairly well, but there was almost constant irregular movement of the trunk.

There was no curvature of the spine. There was no rigidity of the arms; the grasp was good, but there was marked incoordination of both hands on attempting to pick up a penny; the intention tremor was, however, slight. The child was seen again in December, 1902. He had learnt to walk, but the gait was still ataxic. He could sit up, but could not balance himself in a chair without sides. The knee-jerks were brisk; there was no ankle clonus, and the plantar reflexes were flexor in type.

In March, 1903, he had made further improvement, but was still somewhat ataxic. Knee-jerks were present.

In August, 1905, he was again seen. He was now 6 years old, and well grown. He attended an infants' school, and was stated to have normal intelligence. He was still unsteady in walking, but the movements resembled those of chorea rather than ataxia. He could walk quite safely across the room, and could get up from the floor in a normal manner. His mother stated that he could now go upstairs, whereas formerly he had been unable to do so. His articulation was fairly good, but still had a somewhat jerky character. The inco-ordination of the hands was slight. The knee-jerks were obtained; the plantar reflex was indefinite, but was generally flexor in character.

In a lecture published in the *Clinical Journal* (21), I have given a series of cases showing the gradual transition between the condition above described and that of a typical cerebral diplegia with rigid extension of the limbs.

In the discussion which follows I have suggested tha the experimental evidence derived from the ablation of the cerebellum throws considerable light on this subject, for it has been shown by Luciani (I) that animals rapidly recover from the symptoms produced by ablation of the cerebellum, if the cerebral cortex remains undamaged, but if the cerebral cortex be injured in the region of the gyrus sigmoidea, then the symptoms produced by the removal of the cerebellum persist.

This experiment would seem to explain the well-known clinical fact that there may be atrophy, and even complete absence of the cerebellum without any symptoms pointing to cerebellar disease being present. In the class of case under consideration, it would seem probable that there had been some damage to, or lack of development in the cerebellum and cerebrum at or before birth. As the cerebrum becomes developed, so it assumes the co-ordinating function of the cerebellum, and recovery more or less complete takes place according to the condition of the cerebral cortex.

One of the slightest cases which I have had the opportunity of observing was that of a boy, aged 2 years, who was seen in consultation with Dr. Sinclair, of Dundee. The boy began to walk at 18 months, and it was then noticed that he was very awkward in walking. On examination when 2 years old, although he could with accuracy kick a small ball with the right foot, he was unable to do so with the left, and his attempts to do so were markedly inco-ordinate.

GROUP II.—ACUTE ATAXIA (ENCEPHALITIS CEREBELLI).

Under the second heading, " acute ataxia," are arranged a series of cases which may be said to have the following characteristic symptoms.

A child perfectly healthy and of good intellectual development is taken ill with some acute febrile disease, which may assume some definite type, such as measles, or may be assigned to the more indefinite influenza. A period of unconsciousness may be present, but general convulsions seldom occur. The child is kept in bed for a few days and seems to be making a normal convalescence. When, however, the child is sat up in bed, it is found that he is unable to maintain his balance. There is marked inco-ordination of the hands, and, if stood on his feet, he is wildly ataxic. It may be noticed at the same time that his speech is affected. In a few days his general health is restored, but he remains as ataxic as ever. Recovery now gradually takes place, and although in a few cases recovery may be complete in two or three months, in most cases the period of recovery extends over several months, and it may be two or three years.

Such are the main features of the disease, and the following cases illustrate the type :—

Case 3.—G. S., aged 4½, was well till a slight attack of whooping cough in December, 1900. Towards the end of December, she had a disease described as influenza, for which she was medically attended. She was feverish for about a week, and vomited several times. She then developed paresis of the legs and affection of speech, and had also some slight affection of the sphincters. The child had but little headache, and never lost consciousness.

She was the eldest of three children—the other two had whooping cough. The child was first seen on February 7, 1901. She was a well-nourished, healthy-looking child. She could just stand with the legs wide apart, but was unable to walk and tended to fall straight backwards. The gait on attempting to walk was wildly ataxic. There was marked inco-ordination of the hands, the *right* being more ataxic than the left. The grasp of both hands was feeble and ill-sustained.

When laughing, the left side of the face moved better than the right. The ocular movements were good in all directions, except there was some defective upward movement of the right eye. There was no nystagmus, the pupils reacted well to light, and there was no optic neuritis. Articulation was somewhat bulbar in character, but there was no aphasia. The knee-jerks were equal and active; there was doubtful ankle clonus, and the plantars gave a doubtful response. The child was admitted to the hospital, made a rapid and complete recovery, and when seen some three months later was quite well. The plantar reflexes still gave a doubtful response.

It may be argued in this case that lesion was situated in the region of the mid-brain rather than in the cerebellum, as evidenced by the defect in the movements of the face in the expression of emotion, and in the defective upward movements of the eye. The condition of the knee-jerk, the presence of ankle clonus and the plantar response might also be called in support of such a view, and with this view I should be in agreement, but the cerebellar symptoms are produced by involvement of the fibres of the superior cerebellar peduncles in their course toward the red nucleus; and the ataxia may well be considered of cerebellar origin.

Case 4.—A boy, aged 3½ years, developed measles in March, 1904, up to which time he had been perfectly healthy. During convalescence he had an attack in which he vomited, became unconscious and had a series of general convulsions. The stage of unconsciousness lasted for about one week. On April 9, 1904, he was quite conscious, but his arms and legs shook and he was unable to sit up, and could not speak.

The father of the boy is said to have phthisis, and one child in the family has died of tuberculosis.

The boy was first seen on May 13, 1904. He was well nourished and appeared intelligent. He spoke in a slow, hesitating manner. He was unable to sit up in bed without support, and when placed on his legs, walked in the most ataxic manner, flinging his legs about in all directions. There was marked incoordination of both hands, but the grasp was fairly good and equal on both sides. There was no facial weakness, no ocular paresis or nystagmus, and the optic discs were quite normal.

The knee-jerks were active, there was a slight tendency to ankle clonus on both sides, and the plantar reflexes generally gave flexor responses; but on one or two occasions a distinct extensor response was obtained.

Sensation was unimpaired, and all the superficial reflexes were normal.

The mother stated that since his illness the boy has been most untruthful. Not only did he tell lies to obtain his purpose, but he invented stories which could obtain him no possible advantage. His moral sense was perverted.

He was under treatment for some time in the hospital, but made but little progress, and when discharged in July, 1904, was still very ataxic.

He was seen again in November, 1904, and by that time had improved to a slight extent, but was still very ataxic in walking.

In June, 1905, he again came under observation, and considerable improvement had now taken place in his gait and also in the co-ordination of the hands. His articulation was still defective. The knee-jerks were active, and the plantars gave flexor responses. He was seen again in October, 1905, and had improved in every way, and very little manifestation of his former ailment existed.

Another case was that of a child, who was shown by Dr. A. F. Voelcker before the Neurological Society in June, 1905 (*vide* p. 360 of the present volume).

Case 5.—A girl, aged 4¾ years, was supposed to have had an attack of scarlet fever at Christmas, 1904, during which she became unconscious for fourteen days; gradually she recovered consciousness, but has been unable to stand or feed herself since. Her speech was lost, but had gradually returned. There was no vomiting during the initial illness. When first seen, the child was unable to walk or stand, and could hardly sit up in bed. There was also constant tremor of the head. When supported, her gait was markedly ataxic, and at times the progression was cross-legged. There was a tendency to fall backwards. There was marked muscular hypotonus. The knee-jerks were brisk; there was no ankle clonus, and the plantar reflexes were variable, an extensor response being sometimes elicited on the left side. Only slight improvement took place during the two months the child was under observation. When seen in December, 1905, the child showed but little improvement, the plantar reflexes being still variable.

A similar case recorded by Dr. Leonard Guthrie was shown at the same meeting of the Neurological Society (*vide* p. 363).

A girl, aged 7 years, in February, 1904, at the height of an attack of measles, with a temperature of 103° to 104°, developed cerebral symptoms, vomiting, convulsions, unconsciousness followed by squint, retraction of the head, and photophobia. After five days all the acute symptoms subsided, but the patient was aphasic, seemed hyperæsthetic, and rapidly wasted. The optic discs were normal. A month later she was still aphasic, could not sit up or move lower limbs; the knee-jerks were exaggerated, ankle clonus was present, but no Babinski plantar reflex was obtained. There was marked inco-ordination of the upper limbs, and some athetoid movements of the fingers. There was general anæsthesia to pin pricks, but she recognised hot and cold, and appeared to fear pinching. All limbs were slightly rigid. Sphincter control was lost for two months. Then gradual improvement occurred. By the end of May, 1904, she could stand with support, feed herself, and talk.

The child is described as healthy and well nourished. She was mentally slow, but not deficient. Her speech was drawling and bleating in character. Movements of the eyes were normal. There was slight ataxia and tremor of the upper extremities. She could stand alone and steadily when the eyes were closed, but could not walk more than a few steps without guidance. If left alone she reeled, turned half round, and fell backwards or to one side or the other. Loss of equilibrium seemed due to ataxy in placing the feet rather than to vertigo. The gait was ataxic. The knee-jerks were brisk, ankle clonus was present at times. Plantar response was negative or flexor.

The last case in this group which I shall describe is that recorded by Dr. Frederick Taylor (2), and it is of the greatest interest, since it proves that complete and absolute recovery may take place, so that in adult life the patient is able to undertake and perform the usual routine of life.

A boy, aged 4, was admitted to the Evelina Hospital on June 17, 1875. The boy had had whooping-cough three weeks previously. Eight weeks previously he had fallen down three stairs, but did not seem much hurt. For three weeks it had been noticed that his eyes twitched, and he dropped things out of his hand, and was unable to walk. He had vomited after food. He could not stand by himself, and when sitting up he was unsteady, bending slightly forward, and then recovering himself. He could pick up a pin with his fingers. There was occasionally lateral nystagmus. The chief characteristic of the case was the trembling of the limbs and trunk, which had a close resemblance to that of disseminated sclerosis, so much so that the late Dr. Moxon was asked to see the patient, but he believed that the symptoms were due to a cerebellar lesion.

The limbs were quiet as he lay passively in bed, and only trembled to any considerable extent when he sat up or lifted his arms to take hold of an object. Even then the movements were not violent; they were quick and short, but not extensive. The eyes were often examined with the ophthalmoscope, but no change in the fundus was ever seen. During the fortnight after his admission he was several times sick, but the vomiting ceased after June 30. During the next two months there was very little change. On leaving the hospital in September he was said to be better than before. In the summer of 1876 he was said to have much improved in strength, though still suffering from trembling movements.

In February, 1877, he was again seen. He was fairly intelligent looking. On level ground he could get along in a highly ataxic manner. His movements were precisely those of a drunken man. He did not waddle or throw out his legs with the usual ataxia of tabes dorsalis, but having placed one foot on the ground, he could not balance himself sufficiently long on it to put the other in the right place—thus for a few steps he would plunge headlong forward, only just saving himself from a fall, then he would pull up and steady himself. Another moment and he was again staggering forward, frequently crossing one leg in front of the other.

He appeared to have good strength in his legs, for he was lively and active. He seemed in constant danger of falling, but seldom fell. The arms were ataxic. Tremor of the head and neck was scarcely noticeable. His sensation was apparently normal, and he was a lively, cheerful boy. His speech was hesitating and his voice was not properly modulated.

In November, 1878, he was said to have wonderfully improved. His speech was a little imperfect, but he was able to run and to bowl a hoop.

In November, 1904, he was seen again. He was 33 years of age and a clerk. No sign of his former complaint existed; he was an active man, and included hockey among his recreations.

GROUP III.—PROGRESSIVE CEREBELLAR ATAXIA.

Under the third heading, progressive cerebellar ataxia, are arranged a series of cases which may be said to have the following characteristic symptoms:—

A child who has been perfectly healthy and of normal development till a certain age, gradually develops ataxia, which slowly increases so as ultimately to render the patient incompetent to get about. These cases in some points resemble Friedreich's disease, in others Marie and Sanger Brown's hereditary ataxy, but differ from these diseases in many particulars, so that though undoubtedly allied to those conditions, find a place under the present article, not as a separate disease, but as a member of that great group cerebellar and spinal ataxia.

The following case which was under the care of Dr. Voelcker, who regarded it as a case of disseminated sclerosis, was shown at the Neurological Society, June, 1905, illustrates the main features of the disease, and the published cases of Fraser (3), Michell Clarke (4) and Warrington (5) complete the clinical picture.

Case 6.—M. C., aged 11 years, was well till she was 9 years old. It was then noticed that she was losing the power of walking, her arms were becoming weak, and she had difficulty with her speech. The disease slowly progressed, so that eighteen months later she was unable to stand or walk, and unable to feed herself. The impairment in speech had not increased. There had been no vomiting and only occasional headache. She had had some pain in the lower part of her back and in the neck. She was a well-nourished, healthy-looking child of fair intelligence, easily pleased and somewhat emotional. The upper central incisors were notched, and the spleen could be felt below the costal margin, but there was no other evidence of congenital syphilis.

Her articulation was slow and drawling, but all her words were easily understood.

She could move all her limbs well and her muscular power was good. There was no wasting There was a very marked intention tremor of both upper and lower extremities. The child could only stand and walk with support, and even then the gait was wildly ataxic. There was tremor of the head and shaking of the trunk. When at rest there was no tremor.

There was no optic neuritis or atrophy, and no nystagmus. The pupils reacted to light and on accommodation. The knee-jerks were distinctly increased, the arm-jerks were brisk. There was no patella or ankle clonus. The plantars gave most variable responses, more often flexor than extensor; sometimes no response could be elicited. When last seen in December, 1905, she was in the same condition.

Another case is that of a boy who is still under observation :—

Case 7.—H. K., aged 21 (1905), is the second of three children, one brother two years older, and one sister three years younger; both are quite healthy. The father is healthy, the mother was an epileptic and died of general epileptic convulsions when 36 years old.

The boy was like other children till he was 13 years old. He had had convulsions when cutting his teeth, and again had a fit when 4 years old.

When 13 years old he began to suffer from fits, and at the same time it was noticed that he became unsteady in his gait and staggered about. The condition gradually increased, definite epileptic fits occurred at long intervals, but were controlled to a considerable extent by bromide.

During the earlier years of the affection he was under my care as an out-patient, and at that period he would walk into the room with a wildly ataxic gait, so that he seemed constantly in danger of falling; but he never fell. He had most marked inco-ordination of the hands, and an irregular jerky articulation. He was of normal intelligence, and not depressed about his complaint. The knee-jerks were active, there was no ankle clonus, and the plantar reflexes could not be obtained. There was no nystagmus and no spinal curvature or bony deformity of the feet.

His condition steadily got worse, so that when 19 years old he was admitted to the National Hospital under the care of Dr. Ormerod. He was a small lad for his age, and was easily pleased. There was no nystagmus or ocular defect, and the optic discs were normal. There was marked inco-ordination of both arms, so that he could not with any accuracy approximate the finger to the nose. In the legs there was marked inco-ordination, but no wasting or weakness of muscles. The knee-jerks were active and equal; there was no ankle clonus and no response could be obtained from the plantar.

He was seen again some two years later (viz., August, 1905), and he was then in much the same condition, though his gait, if anything, was less ataxic than on the previous occasion. The knee-jerks were still active, there was no ankle clonus, and the plantar reflexes could not be obtained. The disease had made little, if any, progress.

The foregoing cases illustrate three clinical varieties of ataxia which are found in children, and at any given period in the course of the disease, these three varieties may so closely resemble one another, clinically, that it is impossible to distinguish them from one another, and it is only by the history of an acute or gradual onset in a previously healthy child, or a history of congenital weakness, that the varieties can be clinically distinguished. The prognosis in the first two varieties (*i.e.*, the congenital cerebellar ataxia, and the acute cerebellar ataxia) is comparatively good, whilst in the third (progressive cerebellar ataxia) the prognosis is bad.

Instances of the various types above described occur scattered in medical literature under various headings. The cases of congenital ataxia are, for the most part, placed under the heading of cerebral diplegia, or hereditary and family spastic paraplegia, as by Cestan and Guillain (6), who quote the observations of Pelizaeus and Dreschfeld. From the pathological findings in Mlle. Pesker's case (7), since there is affection, not only of the fibres in connection with the cerebellum but also of the cerebrum, it would seem that there is considerable justification for such a classification.

CONGENITAL CEREBELLAR ATAXIA.

The typical case of congenital cerebellar ataxia is one in which the ordinary symptoms of cerebral diplegia do not exist. There is no rigidity of the limbs, but rather hypotonia of the muscles, the knee-jerks are brisk, there is no ankle clonus and no definite and persistent extension of the big toe. The course of the affection is such, that in the most favourable cases almost complete recovery takes place, or more accurately, almost complete normal development takes place. In Collier's article on "Cerebral diplegia" (8), after mentioning the choreic and athetotic movements which occur in these cases, and which he states never improve, he refers to movements resembling disseminated sclerosis, and gives an instance in which such movements slowly developed after pneumonia, in a child aged 7 years; such a case would belong to my third group of ataxia in children, in which, with or without acute illness, symptoms gradually present themselves. Warrington and Monsarrat (5), in their paper on the arrested development of the cerebellum, give an excellent résumé of the subject; their classification is based on the probable pathogenesis, and includes five groups. (1) Complete absence of cerebellum. (2) Cerebellum congenitally small, but histologically normal. (3) General sclerosis, often following acute disease. (4) Atrophy, due to primary vascular disease. (5) Primary atrophy of cerebellar cortex. Although the clinical grouping which I have adopted cannot altogether fit in with the pathological classification of Warrington and Monsarrat, yet it is probable that cases in my group (1) "congenital cerebellar ataxia," correspond with cases in their groups (1) and (2). Cases in my group (2) acute ataxia, "encephalitis cerebelli," correspond with their group (3), whilst cases appear in my group (3), "progressive cerebellar ataxia," which also appear in Warrington and Monsarrat's groups (2) and (5).

It is well known that great deficiency of the cerebellum may exist without any marked symptoms, though, in the majority of cases, motor and mental symptoms occur, and Ferrier (9), in his article on "The Regional Diagnosis of Cerebral Disease," gives a table of cases of cerebellar atrophies from 1879-1895 which bears this out.

No case that I can find, which has been pathologically examined, corresponds, clinically, with the typical case of the first group which I have described, viz., a child who is ataxic from birth, gradually loses the ataxia, and develops, practically, into a normal child. As I have already pointed out, in the case examined by Mlle. Pesker (7), we possess pathological evidence of the condition present in cases of congenital ataxia which do not show any tendency to improvement.

ACUTE ATAXIA.

With regard to the second group, "acute ataxia," this term was first used by Leyden (10) for ataxia of acute onset in a patient, aged 22, in whom, eight weeks after an injury, there was marked ataxia of the limbs, without loss of power or loss of ordinary sensation, but with loss of the appreciation of different weights—a difficulty in speech and attacks of giddiness. The patient did not improve.

Westphal (11) observed cases in which clinical features closely resembled disseminated sclerosis, but with this difference, viz., that the symptoms occurred acutely following on small-pox and typhoid.

Lenhartz (12) described ataxia occurring after acute disease, and gives an account of a boy, aged 8, who, after an acute attack of dysentery, became unconscious and developed acute ataxia, from which he gradually recovered in about one year.

Whipham and Myers (13) describe two cases in which nervous sequelæ, and among them ataxia, followed on variola. They give also a digest of nineteen cases in which nervous sequelæ followed on small-pox or other acute illness; all of these except one occurred in adults.

From the somewhat large mass of literature I have endeavoured to pick out those cases which most closely resemble those described in this paper, with special reference to children.

Shephard's case (14). A child, 5 years old, taken ill at the same time as her sister with scarlet fever, became speechless and ataxic. Recovery gradually took place, so that in fourteen weeks she was again able to walk.

Feith's case (15). A child, 5 years old, developed typhoid, became completely unconscious and recovered after four weeks with aphasia. She then had marked ataxia without any disturbance of sensation, and in three months recovered completely.

Scheper's case (16). A child, 8 years old, developed measles with coma, lasting three days; on recovery there was inability to speak, marked ataxia of the limbs without paralysis, no sensory disturbance and no sphincter trouble. Speech gradually returned, but was nasal and monotonous. The memory was defective. Complete recovery took place.

Hammarberg's case (17). As a child was quite well till 7 years old, when he had cerebral inflammation with cerebellar symptoms. At first there was continuous oscillation in limbs and trunk followed by ataxia. Movements were slow and uncertain. Speech was at first lost, then returned and was indistinct and scanning. This patient remained normal till the age of 24. Then he developed delusions, had fits of mania, had some epileptic attacks and died. Atrophy and sclerosis of the cerebellum was found, with patches of sclerosis in the frontal lobes and in the left olive. Macroscopically, the vermis appeared normal; but microscopically no part was normal, the part of the vermis most affected being the post-lobules. The pia mater was thickened all over the cerebellum.

Spiller (18) gives an account of four cases of cerebellar disease, in three of which the disease was certainly congenital, and the patients remained in a defective condition.

In the fourth case, the following early history is given, which renders it uncertain whether the disease should be regarded as congenital or acquired. The patient was the fourth child of healthy native-born parents. No instruments were used at his delivery, which was at full term. As a babe he had a weak back and did not learn to walk until he was 3 years old. When 5 years old he had an attack of scarlet fever and diphtheria, which interfered with his speech and walking. His gait showed a slight stagger, and the feet were raised very high from the ground. He died when 19 from tuberculosis.

At the autopsy it was found that the corpus callosum was defective, the cerebellum small, and the left cerebral hemisphere atrophied. Sclerosed areas were present in the cerebellum with thickened vessels. Atrophy of inferior olives, and of the middle peduncle of the pons and pontal nuclei, was present.

In discussing the position which this case holds with regard to that described by Marie, Nonne and others, he concludes that it should be regarded as a rare type of that described by Marie, although heredity could not be established.

The paper discusses the relation of cerebellar disease to the symptoms produced, and is furnished with a most useful list of references.

Lüthje (19), in a paper on the acute cerebral and cerebro-spinal ataxia, describes three cases of ataxia following typhoid fever in three children of one family, aged 10, 9 and 6. The course of the disease varied in intensity with these children, being least marked in the youngest, who completely recovered in two months, whereas the recovery of the eldest was not complete in four months. There was a period of unconsciousness from which on recovery there was marked affection of speech, very marked ataxia of the arms and legs, with increased knee-jerks, ankle and patella clonus, but without any disturbance of sensation except stereognostic sense. The other two cases were similar, but the affection of stereognosis could not be demonstrated. Lüthje recognises two forms of ataxia, (1) the pure cerebral form; (2) the cerebro-spinal form; and he lays special stress on the affection of stereognosis as indicative of the cerebral form.

The pathological evidence in support of this form of acute ataxia being due to cerebellar disease is extremely deficient; the case recorded by Hammarberg, and possibly that recorded by Spiller, being the only evidence which can be adduced in favour of this view.

Clinically, those who favour the view that the ataxia is of cerebral origin, bring forward the loss of consciousness, the affection of speech, the mental attitude and the loss of stereognostic sense, which is present in some of these cases, as indicative of a cerebral lesion.

Those who favour the view of the cerebellar origin of the disease point to the character of the ataxia affecting the legs and trunk muscles especially, the absence of loss of power, the hypotonia of the limbs and the character of the reflexes. The affection of speech is rather an affection of articulation than of "speech," and the scanning, monotonous and jerky character may well be the result of defect in co-ordination of articulation. That the initial disturbance is so wide in some cases as to cause loss of consciousness will be denied by none, but it can hardly be used as an argument to prove that the ataxia is of cerebral origin.

To all who study these cases, it is obvious that no line can be drawn separating the cerebellar cases from the cerebral, but in certain cases the cerebellar symptoms predominate, whilst in others the cerebral symptoms are most manifest.

And it may here be well to point out that under the term cerebellar is included not only the cerebellum, but also the cerebellar peduncles in their course to the red nucleus of the opposite side.

What the exact nature of the initial lesion may be still lacks pathological proof. That the lesion is of vascular origin, would seem probable from the sudden onset. Arguing from the analogy of similar cases of acute onset, giving rise to cerebral and cranial nerve symptoms, in which pathological proof is forthcoming, it would seem justifiable to regard these cases as similar in origin to acute anterior poliomyelitis and polio-encephalitis, and this is the view I have taken in a paper published in the *Medical Soc. Trans.* (20). That is to say, the cerebellar symptoms are due to a thrombosis occurring in the finer vessels of this region.

PROGRESSIVE CEREBELLAR ATAXIA.

With regard to the third group, progressive cerebellar ataxia, there is no doubt that these cases are nearly allied to the disease known as "Friedreich's disease," and to the hereditary cerebellar ataxia of Marie and Sanger Brown, the close relationship of which, pathologically, has been shown in a recent paper by Lewellyn Barker (22).

One of the most typical cases of progressive cerebellar ataxia, is that reported by Donald Fraser (3), in a brother and sister. The boy was healthy at birth, and when 3 years old unsteadiness was first noticed, and when 6 years old the reeling was very marked. Up to the age of 30 he was able to walk and do his work of delivering papers. There was no mental unsoundness, but his articulation was slow, gutteral, and hesitating.

This patient died when 33, and the following changes were found: The cortical substance of the cerebellum was little more than half the normal thickness, and there were gaps on the surface, probably caused by collections of fluid in the soft membrane. The white substance presented little apparent reduction, and in proportion to the grey substance appeared of undue thickness. The cells of Purkinje in the cortex were greatly shrunken and contorted, their processes being indefinite and altered in direction. The weight of the cerebellum was 1,260 grains, the average weight being 2,640 grains. The spinal cord appeared normal.

The younger sister of this patient was similarly affected, but the disease developed later and more slowly than in her brother. She could read fairly well. Her walking was so unsteady that she was ashamed to go out. The eyes could only be kept steadily on an object by an effort of will, and they constantly tended to turn diagonally upwards and to the left, and she stated that it was a relief to let them go in that direction. When she lay down she became giddy, and she stated that her eyes ' glisten " ; while this continues she did not dare to move her head, or she would become worse. There was a feeling of nausea, but no vomiting.

One of the best recorded cases in childhood of this type, with pathological investigation by recent methods, is that of Michell Clarke (4), but even this case shows clinically many signs indicative of cerebral disease, and pathologically this is borne out by the marked affection of the pyramidal system.

A boy whose two uncles, aged 12 and 13, were said to have died of the same disease, was quite well till 7 years of age; it was then noticed that he had slight choreiform jerking of the arms and legs. His sight gradually failed. He became unstable, and his speech became affected. He was ataxic in walking and tended to fall backwards. His sight was extremely defective, no change was present in the discs. There was no actual paralysis. The knee-jerks were exaggerated, the plantar reflexes active, and Babinski's sign was present. He gradually got worse, his memory became bad and his speech more indistinct. He walked with an uncertain ataxic gait, and had a tendency to fall backwards. Both legs were rigid and ankle clonus was present. A few months later he was quite helpless, unable to sleep; his legs were drawn up and flexed on the abdomen, and he died about a year after the onset of the symptoms.

At the autopsy the brain was small. There was sclerosis of the occipital region; the cortex was paler and thinner than normal. The cerebellum was rather small, the white matter showed even greater sclerosis than the occipital lobes. Microscopically, the most striking change was seen in the pyramidal system, which was completely degenerated; sclerosis was present in the white matter of the cerebellum and scattered degenerate fibres in the fillets, superior cerebellar peduncles and restiform body.

Numerous instances of ataxia with atrophy of the cerebellum are recorded, but in most of the cases there is present also mental defect, with corresponding change in the cerebral hemispheres, often associated with hydrocephalus.

In conclusion, short mention should be made with regard to three conditions which may give rise to symptoms closely approximating those described in this paper. These conditions are (1) disseminated sclerosis; (2) quiescent cerebellar tumour; (3) past meningitis and hydrocephalus.

(1) *Disseminated Sclerosis.*

It will be readily recognised that the symptoms closely resemble those of disseminated sclerosis, and although in many of the cases the course of the disease is not that which is met with in disseminated sclerosis, yet the clinical features of the disease are so similar that had they occurred in the adult it would be extremely difficult to exclude that diagnosis. Cases of disseminated sclerosis have been described in children both clinically and pathologically, but the pathological evidence is by no means convincing, and, from the pathological evidence collected in this paper, I am inclined to regard these cases as due to cerebral and cerebellar lesion, and not to a disseminated process.

(2) *Quiescent Cerebellar Tumour.*

The second condition which may give rise to symptoms similar to those which occur in the second group, is the presence of a tumour in the cerebellum, and it is well known that in the case of tuberculous tumour of the cerebellum all the acute symptoms may subside, and the child remain in moderate health for months if not for years. Most of these children have, however, very defective eyesight secondary to the optic neuritis and often complete blindness, and in addition usually have some degree of hydrocephalus. In the series of cases under consideration, optic neuritis, secondary atrophy and blindness were not present.

(3) *Past Meningitis and Hydrocephalus.*

That meningitis and hydrocephalus may give rise to the symptoms above described is possible, but the general well-being of the child, the absence of headache, and the absence of any evidence of hydrocephalus from the shape of the head, tend to negative this suggestion.

As with cerebral diplegia, three groups of cases may be recognised:—

(1) Natal or prenatal in origin with some tendency to improve as life advances.

(2) Acquired from acute disease with tendency to improve.

(3) Progressive cerebral degeneration with steady deterioration.

So with the cerebellum a similar classification may be adopted.

The tendency to improvement is however greater, and recovery more complete, whilst the symptoms manifesting degeneration are slower in development in the case of the cerebellar lesion as compared with the cerebral lesion.

SUMMARY.

The object of this paper has been to attempt to classify certain cases of ataxia occurring in children which do not fall under well-known types of diseases.

The cases have been regarded from their clinical rather than their pathological aspect, and from their clinical features it would seem probable that the symptoms are due to lesions of the cerebellum and cerebellar peduncles. This is to some extent borne out by the pathological evidence of reported cases, but there is at the same time considerable evidence pointing to affection of tracts and portions of the nervous system other than the cerebellum.

The cases group themselves into three main divisions:—

(1) Cases in which ataxia has been noticed from earliest life, and in which there is a tendency to gradual improvement (congenital cerebellar ataxia).

(2) Cases in which ataxia has suddenly developed during the course of or after some acute illness in a child who was formerly quite healthy (acute ataxia, encephalitis cerebelli).

(3) Cases in which ataxia has gradually developed in a child who has been healthy and of normal development until the onset of the disease (progressive cerebellar ataxia).

Instances of these various types are given, and it is pointed out that whereas in cases belonging to groups 1 and 2 the prognosis is relatively good, in the cases belonging to group 3 the prognosis is bad.

The pathology of the disease is discussed, and it is shown from the record of the published cases that changes in the cerebellum are the most constant feature in this affection, though such changes are frequently accompanied by other lesions of or lack of development in the cerebrum, pons, medulla or spinal cord.

Further research is needed to establish the above clinical classification on a pathological basis.

A general bibliography will be found in references (5) (18) (19) (23).

Special References.

(1) Luciani. *Il Cervelleto*, 1891. Brain, 1892, vol. xv., p. 291.
(2) Taylor, Frederick. "Case of Encephalitis Cerebelli." *Lancet*, November, 1904, p. 1,416.
(3) Fraser. *Glasgow Med. Journal*, 1880, vol. xiii., p. 199.
(4) Michell Clarke. "A Case of Sclerotic Atrophy of the Cerebrum and Cerebellum." Brain, 1902, vol. xxv., p. 318.
(5) Warrington and Monsarrat. "Arrested Development of the Cerebellum." Brain, 1902, vol. xxv., p. 444.
(6) Cestan and Guillain. *Revue de Médecine*, 1900, vol. xx., p. 813.
(7) Pesker. See *Revue de Médecine*, 1900, vol. xx., p. 832.
(8) Collier. "Cerebral Diplegia." Brain, 1899, vol. xxii., p. 415.
(9) Ferrier. "Allbutt's System of Medicine," vol. vii., p. 366.
(10) Leyden. *Klinik der Rückenmarkskrankheiten*, 1875, Bd. ii., S. 203. "Ueber Acute Ataxie." *Zeitschrift für Klinische Medizin*, 1891, Bd. xviii., S. 576.
(11) Westphal. *Berl. Klin. Wochensch.*, 1872, No. 47.
(12) Lenhartz. *Berl. Klin. Wochensch.*, 1883, No. 21.
(13) Whipham and Myers. *Clin. Soc. Trans.*, vol. xix., p. 164.
(14) Shephard. Quoted by Kühn. *Deut. Arch. f. Klin. Med.*, Bd. 34, S. 77.
(15) Feith. *Allgm. Zeitsch. f. Psychiatrie*, Bd. 30, S. 236.
(16) Scheper. *Berl. Klin. Wochensch.*, 1872, S. 517.
(17) Hammarberg. *Neurolog. Centralblatt*, 1892, S. 142.
(18) Spiller. Brain, 1896, vol. xix., p. 588.
(19) Lüthje. "Die Acute Cerebrale und Cerebro-spinale Ataxie." *Deutsche Zeitsch. für Nervenheilkunde*, 1902, vol. xxii., p. 280.
(20) Batten. *Med. Soc. Trans.*, 1905, vol. xxviii., p. 116.
(21) Batten. "Cerebellar Diplegia." *Clinical Journal*, 1903, vol. xxii., p. 81.
(22) Barker. "Anatomical Report of Two Cases of Hereditary Ataxia." *The Decennial Publications*, University of Chicago.
(23) Nonne. "Anatomischer Befund bei einem Fall von familiärer Kleinhirnataxie." *Arch. f. Psychiatrie und Nervenkrankheiten*, 1905, Bd. xxxix., S. 1,224.

ON THE METABOLISM AND ACTION OF NERVE CELLS.

BY F. H. SCOTT, PH.D., M.B.

Formerly Demonstrator of Physiology, University of Toronto.

With Two Plates.

I.—SOME CONSTITUENTS OF NERVE CELLS AND OTHER CELLS CONTAINING SIMILAR SUBSTANCES.
  (*a*) Introduction.
  (*b*) Nissl Substance and Substances resembling it.
  (*c*) Use of Chromatic Material in other Cells.
  (*d*) Neurosomes and Zymogen Granules.
  (*e*) Nucleus.
  (*f*) Interdependence of the various Constituents.
II.—ACTION OF NERVE CELLS AND HYPOTHESIS OF STIMULATION.

I.—SOME CONSTITUENTS OF NERVE CELLS AND OTHER CELLS CONTAINING SIMILAR SUBSTANCES.

(*a*) *Introduction.*

The presence of blood and lymph vessels in the central nervous system renders it certain that the cells of these organs need nourishment, or, in other words, have a metabolism, and many observers have been able to find differences in different states of this metabolism. Thus, changes have been observed as a result of activity, other changes have been found as a result of injury to the prolongations, and others even through merely removing normal impulses from the cell. These observations, not to mention the various changes known to occur as a result of different intoxications, make it clear that nerve-cells have a metabolism, but they have not cast any light on the nature of the action which is the result of such metabolism. My investigations, conducted chiefly from a histological and microchemical point of view, may perhaps throw some light on this question.

In the year 1899 I (44) published a paper on the microchemistry of nerve-cells, dealing especially with their nuclein compounds. Since that time, although with many interruptions, I have been making a comparison of the compounds found in nerve-cells with those of the cells of other organs. The similarity between the constituents of nerve-cells and those of certain gland-cells, as well as a corresponding difference from those of the cells of other organs, caused me to investigate this subject further.

(*b*) *Nissl Substance and Substances Resembling it.*

The cytoplasm of nerve-cells contains a substance to which prominent attention was first directed by Nissl, and which is commonly called the Nissl substance, Nissl granules, or tigroid. Held (15), who was the first to apply microchemical tests to this substance, came to the conclusion that it is a nucleoproteid. Besides its well-known property of staining with thiazin dyes, Held found that it is soluble in dilute alkalies, leaves a residue on peptic digestion, is not acted on by acids, is insoluble in alcohol, ether or chloroform, and gives no reaction with Millon's reagent or the Adamkiewicz's or xanthoproteic tests. It gave, however, a positive reaction for phosphorus, using Lilienfield and Monti's test. The reactions which I obtained were in the main confirmatory of the above, and where differences are seen they are discussed in my former paper. I found that it contains marked quantities of iron and phosphorus in organic combination, leaves a large residue on peptic digestion, gives a Millon reaction, is insoluble in alcohol, ether or chloroform, but is readily rendered unstainable in thiazin dyes by acids or alkalies through an alteration of its nuclein part. There is no doubt that the conclusion to which Held arrived, that this substance belongs to the class of nucleoproteids, is correct. I shall not go further into these reactions, except as to its relation to acids, which has led to a variance between Bethe (5) and Held. This substance is insoluble in acids, but can be rendered unstainable in thiazin dyes by treatment with acids, which dissolve the iron from all nuclein compounds. As it is the iron-holding part which gives to chromatins their peculiar staining properties, when this has been removed the chromatin no longer stains normally. Aqueous acids, especially hydrochloric and nitric, extract the iron very rapidly. Bünge (7) used hydrochloric acid in alcohol and thought this did not extract the iron in organic combinations, but Macallum (28) has shown this is largely dependent on the amount of material used. As sulphuric acid acts slower and can thus be better controlled, Macallum used alcohol to which 4 per cent. of this acid had been added. This unmasks the iron, that is, changes it from its organic combinations, which give none of the ordinary tests for iron, into an inorganic combination which gives the ordinary tests. The alcohol tends to keep the iron compound from diffusing into the liquid, but even in this fluid if the treatment be prolonged, the iron dissolves out of the tissue. This is especially true of such combinations as occur in the Nissl substances which give up their iron readily, so that if the treatment be prolonged they show an absence of iron, while the chromatin of the nuclei still show a marked reaction. It should be noted, as Macallum (28) has shown, that the above treatment unmasks the iron only in particular combinations. There are many combinations it does not affect. Thus it does not unmask the iron of hæmoglobin or hæmatin. This is also true of the ammonium sulphide reaction for iron, which is, however, a far more general test than that derived from the use of acid alcohols.

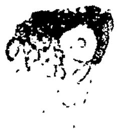
As the function of the Nissl substance is unknown, I thought that an examination of other cells for the presence or absence of a similar substance might cast some light on its use. After first working through the various organs separately, the plan finally adopted was to attach to the same coverslip sections of the same thickness of the different organs which had been treated in the same manner. The sections of the different organs were thus tested at the same time. A number of fixing fluids were tried, but the following were most used: Alcohol in various percentages, Carnoy's mixture, or a solution of sublimate half saturated in water, to which $\frac{1}{10}$ of its volume of formalin, and $\frac{1}{100}$ of its volume of acetic acid were added before use. Toluidin blue with a counterstain of eosin or erythrosin was the combination most frequently employed. They were sometimes applied as Held uses them, but most frequently the sections were first stained in toluidin blue, and then differentiated in dilute erythrosin in water or alcohol. If the thiazin dye were employed alone, the sections were differentiated in dilute acetic acid (1-1000). In employing the reaction for iron a 2 per cent. sulphuric acid in absolute alcohol was used. The sections attached to the cover were left in this fluid for about four hours at 50°, and after washing in absolute alcohol, treated according to Macallum's (29) method in a pure hæmatoxylin solution. After five to ten minutes in this solution the sections were washed in alcohol, and sometimes counterstained in dilute eosin. In digestion experiments, only material which had been fixed in alcohol was employed. The sections attached to the coverslip were digested in a pepsin hydrochloric acid (0·3 per cent.) mixture for a varying time at 38° C, washed and stained in iron-alum hæmatoxylin without any differentiation.

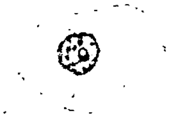
On applying the above methods (stains, solubilities, digestion, tests for iron and phosphorus) to the cells of other organs, I find the only other places where a substance is found in the cytoplasm with similar properties, in quantity comparable at all to that found in nerve cells, are those cells which form the strong proteolytic ferments for secretion, that is, in the chief cells of the fundus glands and in the pancreas cells. In these cells it is confined to the outer striated zone, and to a much less extent in the strands of cytoplasm between the zymogen granules of these cells. The only cells where there was any difficulty in deciding as to the presence or absence of such a substance were those of the serous salivary glands, where basal filaments of a chromatic nature have been described [Solger, Müller, Garnier (12)]. These structures, no doubt, represent a somewhat similar substance, but the amount, under ordinary conditions, is so small that their occurrence has actually been doubted by Maximow (35). By the use of pilocarpine it is possible, as

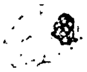
Langley (21) has shown, to produce an outer zone to these cells, and this outer zone then shows the presence in fair amounts of a somewhat similar material, which, however, does not give as marked iron or phosphorus reaction as the substance found in the pancreas or fundus cells.

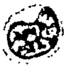
In looking for this substance organs were used from the dog, cat, rabbit, frog and other amphibians, including *Necturus*. In the cells of the liver, spleen, kidney, suprarenal, testis, epididymis, prostate, thyroid, skin glands, mucous glands, muscle or tendon, I have never met with any such substance. Cells containing combinations of mucic or chondromucic acid stain metachromatically with thiazin dyes, and the undifferentiated cytoplasm of these cells may show slight traces of iron, but such cells can readily be distinguished from cells containing a substance similar to the tigroid of nerve-cells. One has only to stain a section from the fundus of the stomach taken in active digestion to see that the chief cells contain this peculiar substance, while it is entirely absent from the parietal cells and other cells of the organ. The figures show the result of treating sections of a few of the organs with some of the methods. As I was looking for a particular substance and not for morphological details, thick sections (about 10 microns) were most frequently used. In such sections it is usually not possible to make out with certainty any details as to the structure of the cytoplasm. The figures with the same numeral were drawn from sections which had been treated at the same time. Thus figs. 1 represent (*a*) a pancreas cell, (*b*) a liver cell, (*c*) a parotid cell, and (*d*) a leucocyte from a lymph gland of the same dog. Figs. 2 are those of a chief cell and a parietal cell from the fundus of a rabbit. Figs. 5 show the distribution of iron in the same cells. Figs. 3 and 6 are from amphibian organs, and show in one case the effect of toluidin blue and erythrosin, and in the other the distribution of iron. Fig. 6*c* is a red blood corpuscle of a frog drawn from a blood-vessel in the same section from which 6*a* was taken. Figs. 7 show the result of digestion with pepsin and hydrochloric acid on sections from the same organs from which figs. 1 were drawn. A nerve cell has been drawn

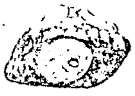
Plate I

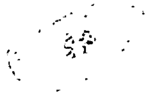

1a

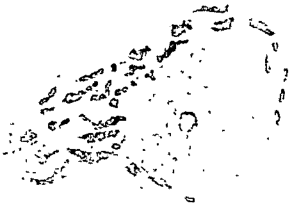

1b

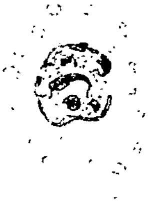

1c.

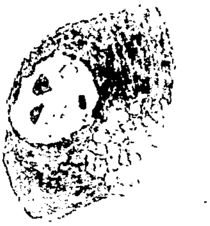

1d

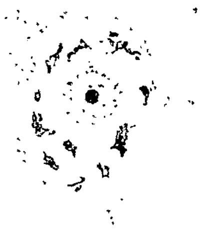

2a


2b


3a

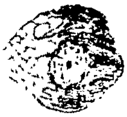

3b




4

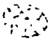
[illegible]

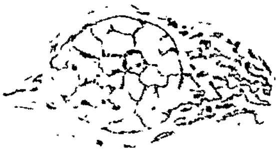
Plate 11



5 a

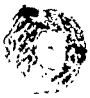
5 b





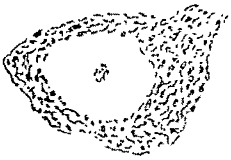
6 a

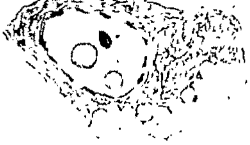
6 c



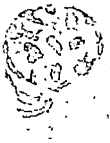
7 a

7 b





8 a



instead of a leucocyte. After digestion there is, in the cytoplasm of all cells, a small amount of undigested material (plastine, Carnoy), but it is a minimum compared with that left in the cells of the three above-mentioned organs. The figures show that this peculiar chromatic substance of the cytoplasm occurs only in the places indicated. It is, however, not merely a case of this substance occurring in the cytoplasm of these cells and in the nucleus in the other cells, for the basichromatin of the nucleus has other properties from this material. Thus it is much more resistant to the action of acids and alkalies, and stains readily with methylgreen, which has, as usually employed, little or no affinity for the tigroid substance of nerve-cells, or the basal region of gland-cells.

I have found only two others who have made a systematic examination of other organs for such a material as the Nissl substance. Both were made some years ago and depended solely on the use of methylene blue. Benda (3) says:—"However, granular structures staining intensely with methylene blue also occur in gland cells, liver cells, pancreas cells, salivary gland cells, tumor cells (sarcomata), and are especially numerous in normal and pathological preparations of blood forming organs, the lymph glands." Eve (11) says: "No substance with the same affinity for methylene blue is present in tendon, muscle, nerves, pituitary body, pancreas, suprarenal, ovary, kidney and thyroid."

The contents of the dog's submaxillary gland and of the chief cells of the cardiac gastric glands, and the matrix of cartilage have a similar affinity for methylene blue, but differ in other respects. The liver cells of rabbit, pig, winter frog and worm commonly contain a granular material, staining similarly with methylene blue.

Benda has evidently not distinguished between the methylene blue staining material of the nucleus and that in the cytoplasm. A material staining intensely with methylene blue is especially abundant in lymph glands, but it is the basichromatin of the nucleus. In the cytoplasm it, at best, would occur only in the basophil granular cells which are very rare. Both of these authors mention the liver cells as containing such a substance. I have repeatedly looked for such a substance, both with methylene blue and other methods, in the liver cells of the dog, cat, rabbit, pig, frog, *Amblystoma*, *Diemycylus*, *Plethyodon* and *Necturus*, and have never found it. The granular material of which Eve speaks might be a part of the second nucleus which is common in liver cells, or it might be due to the staining of the ordinary granules of liver cells by the use of an unsuitable staining solution. By varying the method of use, methylene blue can be made to stain many things of very different nature. An old solution of methylene blue is alkaline, and gives a very diffuse stain. In sections of nervous material, not only does the Nissl substance stain, but the entire section is blue, and it is not easy to differentiate even in alcohol, but may be in dilute eosin or in dilute acids. When properly stained (32) with thiazin dyes, it will be seen that the only other gland cells in which a material with similar properties to the Nissl substance occurs are in those cells in which the proteolytic ferments are formed for secretion, and this distribution may be confirmed by the other tests. As this material occurs only in the outer zone, and to a much less degree in the strands of cytoplasm between the granules, the amount of this substance in these cells varies greatly with the stage of digestion. In the cells of a fasting animal none or very little of this substance is found, because the entire cells are loaded with zymogen granules. In the amphibian pancreas, even after three or four days' rest, there is frequently a little of this substance left. It frequently has a peculiar twisted appearance compared with the remainder of the cytoplasm, and corresponds to the Nebenkern of Nussbaum (39). I shall not go further into the morphology of this Nebenkern, but proceed with the function of the cytoplasmic chromatin.

(c) *Use of the Chromatic Material in other Cells.*

It has been known for some years that the outer zone in some ferment-secreting cells stained with nuclear dyes. Macallum (30), as a result of his observations of such cells in rest and activity, came to the conclusion that: "The chromatin of the nucleus gives rise to a substance which we may call prozymogen . . . and which finally diffuses into the cell protoplasm, uniting with a constituent of the latter as zymogen." Later Macallum (28, 31) showed that this prozymogen, which occupies the outer zone of the cell, contains iron and also phosphorus. Mouret (37) takes a similar view that this chromatic material of the cytoplasm is converted into the zymogen granules, and calls it prezymogen. Similar views as to the use of the chromatic material have been taken by Prenant (43), Mathews (34), Garnier (12), Cade (8), and others. The failure of certain authors, *e.g.*, Pirone (42), to observe the "basal filaments," even in the fundus cells, is due to the use of the triacid staining mixture, the methylgreen of which, in the ordinary solutions, has little or no affinity for nucleins of this class, while it has for the nuclear basichromatin. Figs. 1*a* and 3*c* represent pancreas cells in activity stained with toluidin blue and eosin. Fig. 8*a* shows the hæmatoxylin reaction for iron in such a cell, while fig. 8*b* shows a cell from an animal after four days' fast. It will be seen that as the zymogen granules increase, there is a corresponding decrease in the substance which gives the iron reaction.

In his work on the histology of the gastric glands Bensley (4) confirms the observations as to the presence of such a substance in the chief cells, and mentions methylene blue as one of the suitable dyes to distinguish the ferment-forming cells, on account of its affinity for the prozymogen. He says: "It will be seen from the foregoing that there are two salient features in the structure of the body of the gland, namely, the presence of granules of zymogen in a portion of the cell of varying extent next the lumen, and the presence in large amount in the protoplasm of the outer end of the cell, and in that between the granules, of a kind of chromatin called prozymogen, which stains strongly with hæmatoxylin and other nuclear stains, and gives, after treatment with acid alcohol, a strong reaction for iron." We thus see that nerve cells have in common with these ferment-forming cells a large amount of highly organised iron-holding nucleoproteid in their cytoplasm, the Nissl substance corresponding in its characteristics to the prozymogen of Macallum.

(*d*) *Neurosomes and Zymogen Granules.*

Nerve cells have also the second great distinguishing feature of ferment-forming cells, for they also contain an acidophile granule corresponding to the zymogen granule. Altmann (1) in his "Elementar organismen," described and figured the cytoplasm of nerve-cells with a granular structure like that of other cells. In 1896 Levi (24) by using Galeotti's method (fixing in Hermann's fluid, staining much like Altmann but followed by methylgreen) found an increase of these fuchsinophilous granules after activity. It is to Held (16), however, that we owe an adequate description of the presence and distribution of these acidophil granules in nerve-cells. Held has shown, not only by Altmann's method, but with many other fixing fluids, that these granules which he calls neurosomes are in every nerve and nerve-cell, but are particularly abundant in the axis cylinder end arborisations around the body and dendrites of other nerve-cells. In thin sections it is easy to confirm Held's observations as to the presence and distribution of these granules. In the body of the cell they appear as small round or elongated granules occurring between the Nissl granules in the meshwork of the cytospongium (fig. 4.) In the processes they occur in the meshes of the spongioplasm, and often appear as thickenings of the latter. The neurosomes may be demonstrated with many methods, such as erythrosin and methylene blue, but it is more convenient to use Altmann's method or the bichromate method of Held, which give specific stains to these granules. Altmann's method also gives a specific stain to the zymogen granules of the pancreas—the outer zone being free from any such staining material. If this method merely makes precipitates of proteid from the cell sap, then the granules should also occur in the outer zone as well as in the inner, where they may be seen during life. It is not only by the use of such fixatives and stains that the neurosomes may be seen. They may be seen in isolated cells [Arnold (2)]. Turner (48), by means of a modified methylene blue solution on fresh material, has shown that the protoplasmic processes are surrounded by the ends of collaterals or other nerve branches. These ends contain numerous varicosities which are, I believe, caused by heaps of neurosomes. Held (17) has also shown with methylene blue that these granules are abundant in the nerve endings in muscle.

The properties of the neurosomes and those of the zymogen granules are similar in their main features. The only list of properties of zymogen granules I have been able to find is that of Langley (22), for the granules of the œsophageal glands in the frog. He found the granules were dissolved in acids, less readily in alkalies, in bile almost instantaneously. Alcohol dissolved them partly. They are not soluble in glycerine or in a saturated solution of sugar The granules are not affected by a 5 per cent. potassium or ammonium bichromate solution, but they disappear from the gland cells when small pieces of tissue are left in these fluids. Besides these, they reduce perosmic acid slightly, and are visible in teased preparations. Macallum (28) has also shown they give the ammonium sulphide reaction for iron and contain phosphorus (31). They do not, however, give the hæmatoxylin reaction for iron.

My work was done entirely with the granules of the frog's pancreas, and had the double object of determining their solubility, and of trying to determine why these granules are not preserved in ordinary fixing solutions. Small pieces of fresh pancreas were either left in large quantities of the fluid, or the pancreas was left in a moist place for a couple of hours, and then a small piece laid on the slide and gently pressed with a cover. Large numbers of the granules are forced to the edge of the tissue, and the fluid may then be run under the cover, and the effect watched under the microscope. In sodium chloride (0·6 per cent.) the tissue may be left for four days without any diminution in the number of the granules. Distilled water leads to a disintegration of the granules, but whether there is a real solution is hard to determine. There is a residue left. The same remarks apply to the action of acids which lead to a disintegration. The granules may be left for four days in sodium bicarbonate (2 per cent.), or in lithium carbonate (saturated), or in potassium hydrate (0·5 per cent.). The granules are insoluble in glycerine (four days), but are entirely digested with pepsin and hydrochloric acid. The above reactions, along with those of Langley, show that it is not so much the solubility of the zymogen granules which makes them disappear from the cells in ordinary fixatives, but partly because they are destroyed from osmotic differences, and partly because being so loosely attached to the cytospongium, they are discharged by the contraction of the cells.

The reactions of the neurosomes were determined partly with fresh tissues, but also in material which had been fixed in alcohol. The fresh material was put into the fluid to be tested, and then afterwards fixed in Altmann's fluid or in bichloride. The neurosomes are not soluble in alkalies or glycerine, are altered in acids, but disappear entirely with pepsin and hydrochloric acid. They give, after thorough extraction of the sections (5 microns) first with boiling alcohol and then with ether, a reaction for phosphorus. They do not give the acid alcohol, hæmatoxylin reaction for iron. The ammonium sulphide reaction was not tried. I should add that these reactions were obtained in the neurosomes around the Purkinje cells of the cerebellum.

It is thus seen that the neurosomes correspond in most of their solubilities and reactions to the zymogen granules of the pancreas, and of the œsophageal glands. They perhaps do not reduce perosmic acid to the same extent, and are not as visible in the fresh condition as zymogen granules. It is hard to see the neurosomes in a fresh condition owing to the myelin which tends to make everything homogeneous, but it is probable that it is a lecithin combination which gives to zymogen granules their characteristic appearance, and there is no other organ which contains so much of this substance as the central nervous system The neurosomes can however be seen in isolated cells [Arnold (2)].

The neurosomes are, morphologically, homologous with the zymogen granules of gland cells. It may be, perhaps, a common feature of all cells to store up their products of metabolism in a granular form, and, considering these granules, as I believe most histologists do, as products of metabolism, and not as primary living substance, it is evident that the properties of the granules will be dependent on the characters of the intervening cytoplasm where the granules are formed. When we make comparisons we find that nerve-cells, chief cells of the fundus glands and pancreas cells are classified together and distinguished from other cells of the body by having this intervening cytoplasm impregnated by a similar iron holding nucleoproteid.

(*e*) *Nucleus.*

As the nucleus is the centre of metabolism, no account of the structure of cells would be complete without reference to it, and here again we find these three cells classified together. The character of the nucleus in mammalian nerve-cells is well known. It differs from an ordinary nucleus in containing very little of what is usually termed chromatin. With methylene blue alone only the nucleolus stains, but if a counter stain of eosin be added there is also seen to be a large amount of eosinophilous material—the oxychromatin of Heidenhain. The nucleolus is also seen to have an oxyphile centre. As the oxychromatin stains, although lightly, with acid hæmalums and some other nuclear stains, little difference is seen after these stains between the nucleus of a nerve-cell and those of ordinary cells. The oxychromatin also gives a reaction for phosphorus and a slight reaction for iron, and yet there is a fundamental difference between this substance and the basichromatin of the nucleolus or the Nissl substance, for the oxychromatin is completely dissolved in pepsin and hydrochloric acid, while the basophil material is not. This shows that Mann's (33) objection to the terms "oxy" and "basi" chromatin, on the ground that they are both nucleins, is not justified; for if a nuclein is a compound which leaves a residue on peptic digestion, then the oxychromatin is not a nuclein. The latter remark will also apply to the zymogen granules of gland cells.

The character of the nucleus in pancreas cells varies considerably with the stages of digestion. If one examines the cells in ordinary stages of activity, and there is much prozymogen in the cytoplasm, the character of the nucleus resembles closely that of a nerve cell. All the substance in the nucleus is oxychromatin, except for one or two small masses of basophil material collected around the nucleolus like that of a nerve cell, and perhaps one or two small masses in the remainder of the nucleus (figs. 1*a*, 3*c*). If, however, the cells are at rest, and the cytoplasm is loaded with granules, and there is little prozymogen in the cytoplasm, then the nucleus contains much more basophil material. This relation may be seen in the two figs. 8 which represent the distribution of iron in the two cases. While the nerve-cells of adult mammals and most classes of animals have much nucleoproteid in the cytoplasm and little in the nucleus and thus resemble pancreas cells in activity, it is interesting to note that the Urodela have a reversed condition, and have little or no Nissl substance in their cytoplasm, but much basichromatin in their nuclei [Levi (25), Scott (44)], and thus resemble gland-cells in rest. A similar condition to these Urodela cells is found in the cells of embryo mammals where, as the tigroid develops, there is a disappearance of the primary chromatin of the nucleus [Scott (44)].

The nuclei of the chief cells of the fundus glands of the stomach present the same features as those of the pancreas cells. When there is much prozymogen in the cytoplasm there is little basichromatin in the nucleus but much oxychromatin, and when there is little prozymogen in the cytoplasm there is much more non-digesting chromatin in the nucleus.

(*f*) *Interdependence of the Constituents.*

In the case of these gland-cells we know there is an interdependence between the amount of chromatin in the nucleus, the amount of prozymogen in the base of the cell, and the number of zymogen granules present. These facts may not prove that the iron-holding chromatin of the nucleus gives rise to the iron-holding prozymogen of the cytoplasm, which is, in its turn, metamorphosed into another iron-holding compound—the zymogen granules; but they at least make it probable. In case any one may doubt the value of these microchemical reactions, I may point out that Nencki and Sieber (38), working with gastric juice obtained from a Pawlow's fistula in a dog, confirmed and extended the earlier observations of Pawlow and Frau Dr. Schoumow-Simanowski, that on cooling the juice to zero a fine granular precipitate is obtained. This precipitate, which contains the ferment, contains iron and phosphorus, part of the latter as a lecithin-like body, and part as a nuclein-like body.

A transformation of the Nissl substance into the neurosomes has also been established, or, at least, an interdependence between the amount of the Nissl substance and the number of neurosomes been observed. Activity in nerve-cells leads finally to a diminution of the Nissl substance, although there is probably at first a slight increase in its amount. For some of the recent work on this question see Guerrini (14), Carlson (9), and Holmes (19). Levi (24), on the other hand, has shown that there is an increase in the number of neurosomes (fuchsinophilous granules) during activity, an observation which has been confirmed by Motta-Coco and Lombardo (36). I have made some experiments which will be more fully described later, and, I believe, both of these statements are correct. The anomaly that activity leads to an increase in the number of neurosomes, while in gland-cells it leads to a decrease in the number of zymogen granules, is apparent only because we do not examine a nerve-cell in its entirety, but only the body of the cell, while it is evident that the most important point of activity is the nerve ending where a transference of energy takes place. In the long rest of hibernating animals there is, however, a marked diminution of the Nissl substance [Levi (26), Legge (23)], and corresponding to the disappearance of this substance in fatigue we find also in pancreas cells that excessive activity is also accompanied by a disappearance of the chromatic material in the cytoplasm [Dale (10)].

II.—Action of Nerve Cells and Hypothesis of Stimulation.

If we consider the result of the activities of these three cells we also find there is much resemblance between their actions. Ferments and other catalytic agents have, in all probability, only an accelerating action on changes which ordinarily proceed slowly in the substance on which they act [Ostwald (41)]. The definition of a ferment given by Oppenheimer (40) is: "Eine katalytisch wirkende Substanz, die von lebenden Zellen erzeugt wird und mehr oder minder fest an ihnen haftet, ohne dass ihre Wirkung an den Lebensprocess als solchen gebunden ist; die Fermente sind im Stande chemische Processe auszulösen, die auch von selbst, wenn auch im langsamerem Verlaufe, einzutreten bestrebt sind. Das Ferment selbst bleibt bei diesem Process unverändert. Es wirkt specifisch, d.h. jedes Ferment richtet seine Thätigkeit nur auf Stoffe von ganz bestimmter structureller und stereochemischer Anordnung." An acceleration of changes which ordinarily proceed slowly is also the action of a nervous impulse on another cell. It is also interesting to note that these two forms of activity (ferments and nervous impulses) while ordinarily leading to a decomposition may possibly, under certain conditions, have the opposite effect and lead to a synthetic combination of simpler substances.

Since proteids are the essential part of living matter it is, no doubt, by an alteration of the proteids in that part of the cell where the impulse acts that the whole cell is awakened to activity. There are thus three cells in the body whose function is to control the changes in proteids beyond their own immediate substance, and they not only have this similarity of function, but also similarity of chemical constituents. Do these resemblances mean anything, or is it a coincidence that similarity of function should also be associated with similarity of composition? These similarities have, I believe, a deeper meaning, because all life processes in cells must have a chemical basis, and as all cells in the body are derived originally from one cell, it is probable that cells with similar function would be differentiated along similar lines as regards their chemical constituents. It must be admitted that our methods of distinguishing substances in cells are still very coarse, yet when we do make comparisons with the methods at our disposal we find that the pancreas and its cells, although morphologically resembling the parotid and its cells, has far more chemical resemblance to the glands of the fundus, to which its function has also more physiological resemblance. As the nucleus is the centre of metabolism, it is probable that in all cells it sheds some kind of a chemical substance into the cytoplasm, and as proteolytic and other ferments have been found in all organs, the diffuse blue stain which the cytoplasm of all cells gives with toluidin blue might indicate the presence in slight amounts of prozymogen, although more probably due to an oxidizing or other atom group. On whatever the peculiarity rests the fact remains, that those cells which form and secrete ferments for the destruction of proteids are classified together, for in them an iron-holding nucleoproteid accumulates in large amounts in the cytoplasm. Seeing that nerve-cells have great resemblance both in their chemical constitution and in their action to these cells, and believing that the nervous system is something more than a mere system of conducting paths, I formed the hypothesis that nerve-cells are true secreting cells, and act upon one another and upon the cells of other organs by the passage of a chemical substance of the nature of a ferment or proferment from the first cell into the second; and that a stimulation of the latter is made through a partial destruction of the proteid constituents of the second cell where the impulse acts. The nature of the alteration or destruction produced by this substance is, I believe, very similar to that which occurs in the ordinary digestion of proteids. This hypothesis has to do with the stimulation of cells, and not with conduction which proceeds in the substance between the neurosomes; nor has it anything to do with the ultimate action of the stimulated cell which acts according to its own nature and may, as in muscle, use chiefly carbohydrate. When the impulse reaches the nerve ending it causes, I believe, the discharge of some of the neurosomes which are very numerous in that situation, and which have, I consider, the same relation to the proferment that the zymogen granules in gland cells bear to the proferment of those cells.

In this place I shall not discuss this subject further than to point out that just as irritability and conductivity are not identical so there is, in all probability, a difference between conductivity and the process of excitation of the next cell. Many physiological facts show that although nerve-cells may touch and appear continuous with certain technique, yet they are as distinct centres of metabolism as any other two cells of the body. The action of curari, nicotine or apocodeine, or the fact that a nerve like the vagus or cervical somatic will make physiological connection with the sympathetic, all show the metabolic distinctness of every neurone. Perhaps a fact showing more clearly the independence of nerve-cells is that electrical variations will pass from the dorsal to the anterior root, but will not go in the reverse direction, even if the excitability be heightened with strychnine [Hermann (18), Gotch and Horsley (13)]. This seems to me to be clear evidence that nerve-fibres or fibrillæ are not continuous. The hypothesis of Bethe (6) that the electrical variations increase in the natural direction of conduction, while there is a great decrease in the reverse direction, is negatived by such numbers given by Gotch and Horsley (13). Their numbers show that the conduction is accompanied by the same electrical variation in both directions in the same fibre, but, whenever the impulse passes a synapse, there is a great change. Not only is there this change in the amount of the variation, but there is also a change in the rhythm [Schäfer and Horsley (45)]. This physiological independence of nerve-cells seems to me to show that the process of conduction in one cell to its synapse and the stimulation of the next cell are probably entirely different properties of the neurone. When the changes which constitute conduction reach a synapse it might stimulate the next cell by electrical variations, surface tension changes [Schäfer (46)], or by an alteration in the ionic concentration or other purely physical phenomenon, but the retention and reactivating of such changes is a difficult matter to conceive; and yet the sensations gathered by the sensory nerves are in some manner retained in the central nervous system. Whether the known power of proteids to absorb and retain proteolytic ferments and keep them indefinitely in a passive condition until again brought into a suitable medium, throws any light on the manner in which nerve-cells retain and reactivate the stimuli they have received remains for future investigation.

We thus see that my facts derived from histological study as well as the known facts of neurophysiology, point to a close analogy between the secretion of ferments by the fundus glands and the pancreas and the process of excitation occurring in each synapse of every chain of neurones which is in a state of excitation. Just as in ferment-forming cells we have nucleus, prozymogen and zymogen, so in nerve-cells we have nucleus, pro- or Nissl substance and neurosomes. The process of excitation, like that of secretion, involves, I believe, the discharge of neurosomes in the region of the synapse. Since discharge into other cells means the using up of formed material, it must be an exhaustible process, and the process of complete recovery at the synapse must depend on the integrity of the connection of the synapse with the nucleus and cell body which are the original seats of formation of the material involved in the activity. Of this relation I have experimental evidence, as well as direct evidence, showing the independence between the conducting property of nerves and their power of exciting other cells. These I hope to embody in another paper.

SUMMARY.

A substance of the same nature as the Nissl substance of nerve-cells occurs only in the cells of the pancreas and in the chief cells of the fundus glands of the stomach.

The neurosomes of Held are morphologically homologous with the zymogen granules of gland-cells, and there is an interdependence between the amount of Nissl substance and

the number of neurosomes exactly as there is between the prozymogen of Macallum and the number of zymogen granules.

The nuclei of these three cells also resemble one another.

There is also much resemblance in the action of these three cells in that they all are concerned in controlling the changes in proteids.

On the above similarities the hypothesis was formed that nerve cells also act by a kind of proteolytic ferment.

EXPLANATION OF PLATES.

NOTE.—*All figures were outlined with the aid of a camera lucida. Throughout a Zeiss 2mm. apochromatic objective with occular 4 were employed.*

PLATE I.

Figs. 1.—*a.* pancreas cell; *b.* liver cell; *c.* parotid cell; *d.* leucocyte from a lymph gland. Dog. Alcohol 96 per cent. Toluidin blue, erythrosin.

Figs. 2.—*a.* chief cell; *b.* parietal cell; fundus gland. Rabbit. Alcohol 96 per cent. Toluidin blue, erythrosin.

Figs. 3.—*a.* anterior horn cell, frog; *b.* skin gland, *Amblystoma* larva; *c.* pancreas cell, *Diemyctylus.* Bichloride. Toluidin blue, erythrosin.

Fig. 4.—Purkinje cell, cerebellum, showing the end terminals (filled with neurosomes) of other cells ending around the cell. Dog. Bichloride. Toluidin blue, erythrosin.

PLATE II.

All the cells here represented are stained for iron, except the series, 7*a*, *b*, *c*, and *d*, which have been digested in pepsin and hydrochloric acid, and then stained with iron-alum hæmatoxylin. The colour obtained is usually black: but on this plate all the drawings have been printed in blue.

Figs. 5.—*a.* chief cell; *b.* parietal cell; *c.* mucous cell; fundus gland of rabbit. Alcohol 96 per cent. Acid alcohol four hours and then hæmatoxylin.

Figs. 6.—*a.* anterior horn cell, frog; *b.* skin gland, *Amblystoma* larva; *c.* red blood corpuscle, frog. Bichloride. Acid alcohol 4 hours and then hæmatoxylin.

Figs. 7.—*a.* pancreas cell; *b.* liver cell; *c.* parotid cell; *d.* nerve cell from cortex. Dog. Alcohol 96 per cent. Digested twenty hours in pepsin and hydrochloric acid at 38°. Iron-alum hæmatoxylin without differentiation.

Figs. 8—*a.* pancreas cell in activity; *b.* pancreas cell four days after feeding. *Diemyctylus.* Bichloride. Acid alcohol four hours, hæmatoxylin.

References.

(1) Altmann, R. "Die Elementarorganismen." Leipzig, 1894.

(2) Arnold, J. *Arch. f. mik. Anat.*, 1898, Bd. lii., s. 535.

(3) Benda, C. *Neurol. Central.*, 1895, s. 764.

(4) Bensley, R. R. *Quart. Journ. Micros. Science*, 1898, vol. xli. N.S., p. 361.

(5) Bethe, A. "Allgemeine Anatomie und Physiologie des Nervensystems," s. 142.

(6) Bethe, A. *Op. cit.* (5) s. 344.

(7) Bunge, G. *Zeit. f. Phys. Chemie*, 1885, Bd. ix., s. 49.

(8) Cade, A. *Arch. d'Anat. micros.*, 1901, t. iv., p. 1.

(9) Carlson, A. J. *Amer. Journ. of Anatomy*, 1902, vol. ii., p. 341.

10) Dale, H. H. *Phil. Trans. Royal Soc.*, London, 1904, vol. cxcvii., p. 25.

(11) Eve, F. C. *Journ. of Physiology*, 1896, vol. xx., p. 336.

(12) Garnier, C. *Journ. de l'Anat. et de la Phys.*, 1900, t. xxxvi., p. 22.

(13) Gotch, F., and Horsley, V. *Phil. Trans. Royal Soc.*, London, 1891, vol. clxxxii., p. 495.

(14) Guerrini, G. *Arch. Ital. de Biol.*, 1902, t. xxxviii., p. 247.

(15) Held, H. *Arch. f. Anat. u. Phys.*, *Anat. Abth.*, 1895, s. 396.

(16) *Ibid.*, 1897, s. 204, and *Anat. Abth. Supp.*, 1897, s. 273.

(17) ,, ,, *Ibid.*, 1902, s. 189.

(18) Hermann, L. *Pflüger's Archiv*, 1900, Bd. lxxx., s. 41,

(19) Holmes, G. *Zeit. f. allgem. Phys.*, 1903, Bd. ii., s. 502.

(20) Horsley, V. *Vide* 13 and 45.

(21) Langley, J. N. *Journal of Physiology*, 1879, vol. ii., p. 261.

(22) ,, ,, *Phil. Trans. Royal Soc.*, London, 1881, vol. clxxii., p. 664.

(23) Legge, F., *Monito. Zool.*, 1899. Ref. in *Jahres. Neur. u. Psych.*, 1899, s. 197.

(24) Levi, G. *Riv. di Patol. Nerv. e Ment.*, 1896, vol. i., p. 170.

(25) ,, ,, *Ibid.*, 1896, vol. i., p. 141.

(26) ,, ,, *Ibid.*, 1898, p. 18.

(27) Lombardo, G. *Vide* 36.

(28) Macallum, A. B. *Quart. Journ. of Micros. Sci.*, 1895, vol. xxxviii. N.S., p. 175.

(29) Macallum, A. B. *Journ. of Physiol.*, 1897, vol. xxii., p. 92.

(30) ,, ,, *Trans. Canad. Inst.*, 1891, vol. i., part 2.

(31) ,, ,, *Pro. Royal Soc., London*, 1898, vol. lxiii., p. 467.

(32) Mann, G. "Physiological Histology," Oxford, 1902, p. 215.

(33) ,, ,, *Op. cit.* (32), p. 458.

(34) Mathews, A., *Jour. of Morphology*, 1899, vol. xv. Supp., p. 171.

(35) Maximow, A. *Arch. f. mik. Anat.*, 1901, Bd. lviii., s. 48.

(36) Motta-Coco, A., and Lombardo, G. *Anat. Anz.*, 1903, Bd. xxiii., s. 635.

(37) Mouret, J. *Journ. de l'Anat. et de la Phys.*, 1895, t. xxxi., p. 221.

(38) Nencki, M., and Sieber, N. *Zeit. f. Phys. Chemie*, 1901, Bd. xxxii., s. 291.

(39) Nussbaum, M. *Arch. f. mik. Anat.*, 1882, Bd. xxi., s. 296.

(40) Oppenheimer, C. "Die Fermente und ihre Wirkungen," Leipzig, 1903, p. 18.

(41) Ostwald, W. *Verh. d. Gesell. Deutsch. Natur. u. Ärzte*, 1901, Bd. lxxiii., Abt. 1, s. 184.
(42) Pirone, R. *Zeit. f. allgem. Phys.*, 1904, Bd. iv., s. 62.
(43) Prenant, A. *Journ. de l'Anat. et de la Phys.*, 1898 and 1899.
(44) Scott, F. H. *Trans. Canad. Inst.*, 1899, vol. vi., p. 405.
(45) Schäfer, E. A., and Horsley, V. *Journ. o Physiology*, 1886, vol. vii., p. 96.
(46) Schäfer, E. A. *Brain*, 1893, vol. xvi., p. 134.
(47) Sieber, N. *Vide* 38.
) Turner, J. *Brain*, 1901, vol. xxiv., p. 238.

THE ONSET OF HEMIPLEGIA IN VASCULAR LESIONS.

BY A. ERNEST JONES, M.D., M.R.C.P.

Assistant Physician to the Dreadnought Seamen's Hospital.

INTRODUCTION.

THE studies on which this paper is based owe their initiation to a suggestion of Sir William Gowers, to the effect that the *post-mortem* records at University College Hospital should be investigated with a view to ascertaining the facts as to the occurrence of the different varieties of hemiplegia in the sleeping or waking state. In the course of the work it has seemed to me worth while to analyse other points noted in the records; some of the results thus obtained will be here incorporated and compared with conclusions based on other sources of information presently to be referred to. The precise clinical diagnosis of the vascular lesion in a given case of hemiplegia thus brought about is rarely a certain one, even yet; this diagnosis is a necessity before a reliable prognosis can be given, and also yields valuable indications as to treatment. Its practical importance is evident when one recollects that one-twentieth of all the deaths in this country last year were due to such lesions.

METHOD.

The points of value in the diagnosis of a recent case of hemiplegia are not very numerous, and to elucidate the truth as to their significance it is useless to refer, as is often done, to clinical experience alone. There is but one method, namely, to study statistically a large and consecutive series of cases that have been investigated before and after death. Such opportunities of personal investigation are rare, at least as far as an adequate series is concerned, so that I have been impelled to work along the lines indicated in this paper.

A knowledge of the numerous fallacies to be avoided has necessitated such pruning of the material that the results attained seemed very disproportionate to the labour expended; they are, however, perhaps worthy of record for the assistance of future workers.

I feel the necessity of prefacing a few remarks on the study of hospital notes, for it is a method of study that has undeservedly fallen into discredit. This has come about because it has been generally recognised that it is one which is singularly open to fallacy; these fallacies are not, however, inevitable, as is often supposed, and after their careful avoidance this method may yield information of considerable value and otherwise inaccessible. A few words, therefore, will not be out of place here on the nature of these fallacies. They fall into two main groups: *first*, those due to the selected nature of the material under consideration, and, *secondly*, those due to errors in the process of recording the facts. Such errors may come from one or more of three sources; first, the failure to assign proper evidential value to each statement in the patient's history; secondly, the various mistakes which are possible in direct observation of physical signs; and, last, the erroneous interpretation of these observations. It is unnecessary to elaborate these latter points further here, though it might be well to insist on the importance of the last source of error, especially in the influence that it exerts on the actual observation and description of physical signs. A crude instance of all three fallacies would be the following case. A patient says that he has never had a paralytic stroke, though twelve months ago he had a bad fainting attack; some stiffness of the right arm only is found, and is ascribed to senile osteo-arthritis, the characteristic signs of the latter affection being described; after death a small clot is seen pressing on the left internal capsule.

The sources of fallacy that may be placed in the first group are, on account of their subtlety, more often overlooked. Consideration of any such material as the present series of cases at once shows that it has undergone a number of selections through various influences. Thus, before drawing any conclusion on a given subject, as for instance the age at which cerebral hæmorrhage most often occurs, we have to remember that we are dealing, not with a consecutive series of cases of cerebral hæmorrhage, but with only those cases of this lesion that present hemiplegia as a symptom; not even with a consecutive series of hemiplegias due to cerebral hæmorrhage, but with hemiplegias due to this lesion that die, and which die in a busy hospital. Further, the conditions that influence admission to this hospital are numerous, such as proximity of dwelling or employment, rank in life (which in the lower classes is often a question of age as many of the oldest people tend to drift to the infirmary), time of day or night, &c. So that in stating any conclusion it is essential to find out first how it may have been affected by any of these selective factors. In many instances these factors may be directly measured, in others their influence may be estimated more or less approximately; when this cannot be done, they must be so incorporated in such a manner as to qualify the conclusion formulated.

MATERIAL.

A study has been made of all the cases of hemiplegia that died in University College Hospital between the dates of its foundation and the accession of the present staff. This period extends over about sixty-five years, during which time there were in use about seventy beds for adult medical cases. The cases selected have been those of vascular lesion only. Other causes of hemiplegia, such as tumour cerebri, traumatic and inflammatory lesions, have been excluded, as have also cases where the *post-mortem* diagnosis was unsatisfactory or indeterminate, such as local cerebral atrophy, cerebral sclerosis, cerebral congestion. In all there are left 160 lesions, which occurred in 139 patients; 123 were cases of hæmorrhage, 24 of thrombosis and 13 of embolism. These numbers of course in no wise indicate the actual relative frequency of the conditions mentioned, a problem which I do not intend to discuss at present. The fewness of the cases is a little remarkable in a hospital having a rapid transfer of patients, and is in part due to the number that had to be excluded. This illustrates the very great difficulty that exists in taking notes which will be useful for research, unless the questions raised by the research are known beforehand and therefore specially investigated.

OCCURRENCE OF THE ACCIDENT.

(1) *Relation to Exertion.*

It is now recognised that it is exceptional for any of these lesions, even hæmorrhage, to occur during great exertion, and that the importance formerly attributed to this factor was much exaggerated. In only one or two of the cases now under consideration was the lesion preceded by any severer exercise than that involved in walking the street. Nor do we find that this was so in more than ten of Gintrac's 840 cases (1), recorded between fifty and a hundred years ago, at a time when clinicians were especially possessed with the importance of this factor. Natural acts involving effort, such as defæcation and coitus, used to be regarded as much more dangerous in this connection than they are now held to be. Neither was mentioned as having any relation to the onset in the present cases. On an average, ten minutes of the twenty-four hours are spent under such circumstances as might lead to an attack of cerebral hæmorrhage that occur in these ten minutes being attributed to the act of defæcation; ten minutes being a hundred and forty-fourth part of twenty-four hours, then at least five of Gintrac's 840 cases should have been so attributed, even if such attacks occurred under these circumstances no more frequently than at other times. But, in fact, only two of his cases are said to have been brought on by defæcation, and these were specially recorded on account of this feature; so was also the only attack occurring after coitus, an attack that took place on the patient's wedding night. Similar comment applies to the factor of mental excitement, which was noted in only nine of Gintrac's cases, and in none of the present series.

But if it does not seem necessary often to invoke severe exertion as the determining cause of cerebral hæmorrhage in a patient whose miliary aneurism is approaching the bursting point, there is on the other hand some evidence, as we shall see when dealing with the subject of rest and sleep, to shew that in such patients the variations of blood pressure that obtain in normal life are sufficient in themselves to precipitate the grave event. Sudden change of posture may be of importance in causing such variations in patients whose compensatory vaso-motor mechanism is failing. And there is in this series a considerable group of cases of hæmorrhage with a history to the effect that the stroke took place within a few minutes of getting out of bed.

(2) *Relation to Rest.*

If these lesions occurred with equal frequency during rest and customary exertion, and if the particular patients spent as much time in bed as other people, then we might expect to find that about a third of the attacks took place in bed. The facts are these:—

	Number of cases with point noted.	Percentage occurring in bed.
Hæmorrhage	114 ...	18·4
Thrombosis	22 ...	31·8
Embolism	13 ...	54·6

Now, are there any reasons why any of these lesions should enter this series in such a way as to raise its "bed percentage?" There are excellent grounds for believing that hæmorrhage kills much more quickly than either of the other two, and we may suppose that cases of rapidly fatal hæmorrhage occurring in bed at night would be less likely to be brought to the hospital than similar cases taking place in the daytime, while, for instance, the patient was at work. But the number of cases that are fatal in a few hours is very small, so that we need raise our bed percentage for such cases only by a little, say, to 20 per cent. The next possible fallacy is the likelihood of any of these cases occurring in individuals who spent more time than usual in bed, *i.e.*, who were invalided from some cause or other. As regards hæmorrhage, this was operative to a certain extent. Of the ten attacks of hæmorrhage that occurred in hospital, five did so while the patient was in bed. Again, of the twenty-one cases in which hæmorrhage took place while the patient was in bed, five had suffered from a previous attack of hæmorrhage and one from cerebral thrombosis. Some of these previous attacks had occurred years before, and these would incapacitate to a less extent than more recent ones. Still, on the whole, we may assume that quite 40 per cent. of the twenty-four hours were spent in bed by the hæmorrhage patients, taking 30 per cent. as the normal proportion. And as only 20 per cent. of such attacks took place there, this definitely suggests that rest in bed to some extent protects against hæmorrhage.

Taking now *thrombosis*, the average age of these patients was less than that of the hæmorrhage ones, and there had been no prior attack or immediate illness in any of the cases, so that the time spent in bed by such patients was approximately normal. As less than a third of the thrombosis attacks took place during rest in bed, we have then no reason to suppose that such rest makes thrombosis either more or less likely to happen than moderate exertion.

As regards *embolism*, most of the patients had, of course, heart disease, a potent cause of confinement to bed. All the four embolisms that occurred in the hospital occurred while the patient was in bed, thus illustrating this point. With the limited data at our disposal, it is impossible to estimate this factor, so that it is also impossible to comment on the relation of rest to embolism

(3) *Relation to Sleep.*

The amount of time spent in sleep is on the average greater in persons who spend more time than usual in bed, though how much so we do not know. We must, therefore, consider the subject of sleep in relation to that of rest in bed.

In the present series there are :—

	Number of cases with point noted.	Percentage with onset during sleep.
Hæmorrhage	110	9·9
Thrombosis	22	22·7
Embolism	11	27·3

In other words, thrombosis occurred during sleep more than twice and embolism nearly three times as often as hæmorrhage. Comparing this sleep incidence with the bed incidence, we note that of the hæmorrhage and embolism attacks that occurred in bed one half did so while the patient was awake, whilst this was only true of one-third of the thrombosis attacks. What, now, is the proportion of time that similar patients usually spend awake in bed?

Dealing first with *hæmorrhage*, we have to remember at the start that the sleep incidence would doubtless be raised could we include the cases which die at home during the night, though, as we remarked above, this increase would probably be but slight. We also saw that patients in whom hæmorrhage occurred probably spent rather more time in bed than those who suffered from thrombosis, though less than those in whom embolism occurred. They would differ from the instances of embolism in sleeping rather more than normal individuals, for one of the causes of confinement to bed was a previous hæmorrhage, and this, when recent, would be likely to bring about stupor. On the whole, we might expect that they slept for quite three-quarters of the time they spent in bed, altogether then for about 30 per cent. of the twenty-four hours. Yet we find from reference to the above table that, instead of 30 per cent. of the attacks occurring during sleep, less than 10 per cent. did so. This strongly suggests that sleep protects against cerebral hæmorrhage to an even greater extent than rest in bed. This protection that sleep seems to afford is illustrated by the facts that of the ten attacks that took place in the hospital only two came on during sleep, and of the fourteen cases of repeated attacks only one occurred during sleep. Different authorities usually state either that the occurrence of cerebral hæmorrhage during sleep is very rare, or that it is very common. On the one hand, Dana (2) says that if a person finds that he is hemiplegic on waking in the morning we may assume pretty safely that the lesion is thrombosis and not hæmorrhage, as supposed cases of hæmorrhage occurring during sleep usually turn out to be thrombosis. On the other hand, Charcot (3) says that in infirmary work he found that hæmorrhage most often occurred, not merely in bed, but during sleep and at night. Sormani (4) held the same opinion, but in this instance it was not founded on cases examined after death. Vlantassopoulo (5) summarises three factors in his statement that hæmorrhage occurs "either during sleep, or more exactly at the moment of waking, or else in the first hours that follow rising, this being perhaps due to the changes in the hydraulic conditions of the cerebral circulation." Thevenet (6) says that this statement is endorsed by most clinicians to the extent of being supposed to possess some diagnostic importance. Such contradictory statements, based on no consecutive series of cases, show the danger of generalising from personal impressions. The only consecutive series I know of is Gendrin's (7). Out of 176 cases in which cerebral hæmorrhage was found *post mortem*, in 97 (or 55 per cent.) it had occurred during sleep, and in 84 of these during the night sleep. The great divergence of the present results from those of Gendrin appears in several of the points investigated, and cannot be explained completely, even by the selection of his cases from the aged infirm. My results harmonise better with those of Tooth (8), who found that of 246 attacks of hemiplegia in 19·1 per cent. the onset was during sleep; unfortunately, the diagnosis of the lesion was not verified in these cases, and, no doubt, in a number of them it was thrombosis.

Turning now to *thrombosis*, we saw that there was no reason to suppose that these patients differed much from normal people in the time they spent either in bed or during sleep. The figures then suggest that sleep did not in itself make thrombosis more likely to occur; indeed, as far as they go, they point rather in the other direction. This is not necessarily antagonistic to such statements as that of James Taylor (9), to the effect that one of the commonest conditions under which thrombosis occurs is during profound sleep, for he refers in this connection to very old people who have recently suffered great fatigue, and the present series of thrombosis cases was not composed of the senile invalids commonly taken to be the kind of patients who suffer from this affection.

As regards *embolism*, the evidence is much more equivocal, on account both of the smallness of the numbers and the unknown influence of confinement to bed. In spite of this confinement, heart cases are not notoriously good sleepers, so that we might expect quite half of the time spent in bed to consist of waking hours. The figures thus give no reason to suppose that sleep predisposes to or protects against embolism. Of the four attacks of embolism that occurred in the hospital only one did so during sleep.

(4) *Time of Onset.*

On this subject also very contradictory assertions have been made. Charcot (3) said that *cerebral hæmorrhage* occurred most often in the late hours of the night; he gave no figures in support of this. Sormani (4), basing his statement on several thousands of cases, said that there were two especial times of the day when the accident occurred, one between two and four in the morning, the other between three and five in the afternoon. I suspect, however, that these cases were obtained from the official returns of the Italian Registrar-General, to whose department Sormani belonged, and that the lesions were not verified. Dana (2) says that morning and evening are the times of election, the accident being rare in the middle of the day. On the other hand, Gendrin (7) gives details of the precise time of ninety-three cerebral hæmorrhages inspected after death, and in this series nearly two-thirds of all the attacks took place within four hours, namely those between midday and two p.m. and midnight and 2 a.m.; in other words, the precise opposite of the times given by the other authorities. Gendrin's results are most extraordinary, and are not confirmed by any other observer; they serve, however, to cast doubts on statements based on less exact data. Of the present series the exact hour was given in sixty-two instances, and the approximate hour in forty-two, making 104 in all.

	Gendrin.	Mushet.	Present Series.	Total.	Percentage.
Noon to 2 p.m.	26	1	9	36	19·8
Midnight to 2 a.m.	31	1	2	34	18·7
2 to 7 a.m.	8	1	11	20	10
7 to 12 a.m.	5	0	16	21	11
2 to 8 a.m.	9	3	25	37	20·3
8 to 12 p.m.	14	1	19	34	18·7
Total	93	7	82	182	

In the above table Gendrin's series is given, together with the present one, and a small series of Mushet's (10); all the cases were verified after death.

If we consider the present series first, of ninety-two cases only nineteen occurred at night (between midnight and seven a.m.), while seventy-three occurred in the other hours; in addition, two cases occurred in the "morning" and ten in the "evening," so that the preponderance of day over night is even more marked than appears in those figures. Half the cases occurred between ten in the morning and five in the afternoon, a seventh between five and eight in the morning, an eighth between nine in the evening and midnight, and a tenth between six and eight in the evening. Four-fifths of the cases, then, occurred in the first three of these periods, namely, at midday, in the rising hours of the morning, and in the late evening, which would seem in these instances to have been the elective times. The purely relative value of these data, however, is shown by a glance at the accompanying table, which reveals the entire discordance between my results and those of Gendrin. Thus, of his cases over 60 per cent. occurred in the four hours following midday and midnight, whereas only 13 per cent. of the present cases did so; again, only 24 per cent. of his cases occurred between two and twelve p.m., whereas 54 per cent. of the present cases did so. The chief conclusion to be drawn then is that definite statements such as Dana's and

Charcot's must be regarded with some doubt. The onset would seem to be determined by such variable factors that the time of day is in itself of little interest, as is the time of year, although much significance has been ascribed to both of these.

As regards *thrombosis* and *embolism* similar remarks apply, but I will give shortly the main facts gleaned from this series. The time of onset was stated in twenty cases of thrombosis and thirteen of embolism, precisely in four of these and eight of those. In both instances nearly half the attacks occurred in the middle of the day (between nine a.m. and three p.m.), about the same proportion as in those of hæmorrhage. But, whereas only 11 per cent. of the hæmorrhage attacks were in the early morning (between one and six), in thrombosis and embolism the percentage incidence was 30 and 38 respectively. The tendency to dwell on the importance of the early morning in relation to occluding lesions should be checked, however, by the knowledge of the limited extent of the data.

THE SYMPTOMS OF ONSET.

The precise initial symptom was not stated often enough in such a manner as to render a summary of the records of any value on this point. In a general way it may be said that they strongly confirmed the current teaching as to the frequence of nausea or *vomiting* at the onset of cerebral hæmorrhage, and its comparative uncommonness in other vascular lesions, a fact first insisted on by Sir William Broadbent (11) thirty years ago. Twitchings and *convulsions* were commoner with the occluding lesions than with hæmorrhage, and were more often met with when the cortex was affected. General convulsions occurred in nearly a third of the embolism cases; this was so within twenty-four hours in twenty-four out of ninety-seven cases recorded by Eliza Walker (12), but in only seven out of seventy-two recorded by Newton Pitt (13). The last observer's series included an unusually large number of latent embolisms, which the other series did not. Convulsions may occur in cerebral hæmorrhage apart from either meningeal or intraventricular bleeding, though they are more frequent under these circumstances. Of thirty-six cases of intraventricular hæmorrhage recorded by Thomas Jones (14), there were convulsions in four only, which accords with the present observations. Hirtz recorded seventy-seven cases of hæmorrhage that showed convulsions, and in fifty-five the blood was intraventricular.

Loss of consciousness is important in that often it is regarded, whether rightly or wrongly, as one of the main aids to diagnosis. For instance, Trousseau (15) attached such value to this that he asserted that preservation of consciousness at the onset of the attack excluded all idea of a hæmorrhage. Jeannin (16) similarly lays down that there is *always* loss of consciousness with cerebral hæmorrhage. Yet we have Leandier (17), in his article on cerebral hæmorrhage in Bernheim's System saying that the apoplectic onset of this affection is less frequent than a gradual onset with no loss of consciousness Again, Vlantassopoulo (18) insists that the cases that recover have for the most part not been accompanied at the onset by the least trace of loss of consciousness.

It is not common to get an accurate description of the initial symptom from a bystander, but it was mentioned as consisting in immediate loss of consciousness in two cases which did not prove to be primary intraventricular hæmorrhage, as cases showing loss of consciousness as the initial symptom are usually said to be. It is well known how emphatic Trousseau's (19) dictum was on this point, namely, that such descriptions if carefully investigated *invariably* reveal some earlier symptom than loss of consciousness.

I shall consider here only the subject of loss of consciousness occurring within a short time of the onset; terminal stupor and coma are, of course, almost invariable, which is one of the reasons why ingravescent coma of late onset is of such sinister omen.

(1) *Relation to Lesion.*

It seems to be generally accepted that *occluding lesions* cause loss of consciousness less frequently than does hæmorrhage, and that such loss is less profound and lasts for a shorter time. This statement, first ascribed to Todd, and emphasised by Lancereaux (20), is repeated by Nothnagel (21), Monakow (22), and most writers on the subject. Saveliew (23), on the other hand, in an exhaustive work based on 191 cases of cerebral embolism, says that *most* of such patients lose consciousness, though often for a short time only, and that preservation of consciousness is relatively rare. I do not know of any series in which have been systematically investigated the intensity and duration of the coma, or the correlation of the phenomenon with the intensity or even the existence of paralysis. Some facts bearing on the frequency of coma are given here. Consciousness was lost in ten out of twenty-two cases of *thrombosis*, and in six out of thirteen of embolism; in neither instance did this loss bear any relation to the site or extent of the lesion. Of thirty-four cases of *embolism* collected by Gintrac (24), eleven showed loss of consciousness. Of sixty collected by Thevenet (25), twenty-one showed it; of these twenty-one, eight were carotid or basilar embolisms, and two more were cases of simultaneous blocking of both Sylvian arteries, so that only eleven out of fifty single embolisms in arteries other than the carotid or basilar caused loss of consciousness. Incidentally this emphasises the importance of these two arteries in relation to this symptom. Newton Pitt's (13) four cases of basilar embolism showed no symptom except loss of consciousness. Consciousness was lost in twenty out of thirty-three cases of embolism collected by Lancereaux (26), and clouded in several others. It was lost in three out of Jeannin's seven cases, and in forty-five out of sixty-four cases of Eliza Walker (12), being slightly impaired in eight more of these last. Of the total, then, of *201 cases* of embolism, consciousness was lost in at least 47·7 per cent., while it was affected in nearly 60 per cent. This tallies, as it happens, with the figures relating to embolism given in the following table.

Turning now to the subject of *cerebral hæmorrhage*, we find that consciousness was lost early in eighty-nine of 111 cases in the present series, *i.e.*, in over 80 per cent. This agrees fairly well with the figures given by Thomas Jones (14), 72 per cent. of thirty-six cases, and Gintrac (27), 69·3 per cent. of eighty-eight hæmorrhages into the brain substance; there was loss of consciousness in only 38 per cent. of the purely cortical hæmorrhages described by the latter writer. Of these 235 cases taken together, consciousness was lost in nearly 75 per cent. In the table of the cases included in the present series, I have considered hæmorrhage under two headings, intraventricular and extraventricular; the former was nearly always secondary. It is worthy of note that consciousness was retained till near the end in four cases in which there was *post-mortem* evidence that a considerable quantity of blood had been present in the ventricles for some time. One of these patients lived for five days. There is a case of Charcot's (28) on record where the patient recovered and lived for six months in this condition, and Rokitansky has published similar cases.

	Number of Cases.	Percentage with loss of consciousness.
Ventricular Hæmorrhage ...	55	85·5
Non-Ventricular Hæmorrhage ...	49	69·4
Thrombosis	22	45·5
Embolism...	13	46·1

It is likely that the percentage is heightened a little by the fact of the cases being taken from the records of a busy hospital to which unconscious patients would be likely to be brought. The figures illustrate the two ways in which a clinician may be misled by attaching undue importance to the phenomenon, namely, by overlooking the existence of the following two classes of cases. On the one hand, there is a large group—certainly more extensive than shown in this table—of cerebral hæmorrhage cases having the onset typical of thrombosis, and on the other, there is a large group of thrombosis cases the onset of which is apoplectiform. This latter group, first recognised by Rochoux in 1812, has been carefully described by Durand-Fardel (29) in his monograph based on 181 cases of cerebral softening. He says that 58 per cent. of his cases presented this mode of onset, a considerably higher proportion than is seen in the series now recorded. There is no exact evidence as to which of the above groups is the larger; Tooth (30) refers to the former as being so. Some writers still maintain, as Copland (31) insisted, that the onset of paralysis, followed later by an apoplectic attack, means thrombosis. As a matter of fact, however, there is a special type of cerebral hæmorrhage of which this occurrence is equally characteristic; similarly ingravescent apoplexy occurs at times with thrombosis, and not only with hæmorrhage as Copland taught.

It will thus be seen that our positive knowledge on this subject is not great, and a series would prove of great interest in which was studied accurately the incidence of the phenomenon and its relation to such features as the intensity of the paralysis, the extent and site of the lesion, &c.

(2) *Relation to Age.*

Durand-Fardel (29) said that the apoplectiform variety of onset of thrombosis was the more likely to occur the older the patient. In his series, of twenty-nine such attacks only eleven were below sixty and one below twenty, whereas of thirty-four "gradual" onsets twenty-four were below sixty, and six were below twenty. Brissaud (32), on the other hand, says that it is in senile softening that the progressive, gradual onset is seen most often. The present series, although only a little more than a third of Durand-Fardel's in number, as far as it goes tends to support Brissaud's view. The following table is based on eighty-five cases.

		Percentage of cases below 20.	Percentage of cases above 60.
Apoplectiform Onset	Durand-Fardel ...	3 ...	62
	Present Series ...	0 ...	18
Gradual Onset	Durand-Fardel	18 ...	29
	Present Series	9 ...	27

(3) *Relation to Survival.*

Coma supervening at a late date is always of bad import; it would seem, as shown in the accompanying table, that hemiplegia of apoplectiform onset, whatever its cause, carries with it a similar increase in the gravity of the prognosis. Of the commoner variety of hæmorrhage—the extraventricular form—more than twice as many patients died of the immediate attack when the onset was apoplectiform than when it was not so.

	No. of Cases.		Percentage dying within 24 hours.	Percentage dying within a week.	Percentage dying within a month.	With loss of consciousness. With no loss of consciousness.
Intraventricular Hæmorrhage ..	55	47	66·0	92·0	98·0	With loss of consciousness.
		8	25·0	75·0	100·0	No loss of consciousness.
Extraventricular Hæmorrhage ..	49	34	17·6	64·7	88·3	With loss of consciousness.
		15	13·3	33·4	40·0	No loss of consciousness.
Thrombosis ..	22	10	10·0	30·0	70·0	With loss of consciousness.
		12	9·3	25·0	58·3	No loss of consciousness.
Embolism ..	13	6	0	50·0	50·0	With loss of consciousness.
		7	0	14·3	14·3	No loss of consciousness.

LENGTH OF SURVIVAL.

(1) *Relation to Lesion.*

There can be little doubt but that the immediate prognosis is much graver with *hæmorrhage* than with the other two lesions, though it is an exaggeration to say, as Brissaud (33) does, that early death is infinitely rarer with softening than with hæmorrhage. As so many of the patients of the present series were admitted for the actual attack, one cannot ascertain directly the absolute frequency of recovery from these lesions; this is shown by the surprising number of hemiplegics of twenty years' standing that may be seen in our infirmaries, while of the present series no case dated further back than five years. Dana (34) says that two-thirds of patients with cerebral hæmorrhage recover from the immediate attack, which may, I suppose, be taken to last a fortnight. On the other hand, 72·5 per cent. of 109 such patients in the present series died within a week. This discrepancy may be explained in part by the fallacy above mentioned, and in part by the fact that in the period in which the attacks occurred, the mortality from cystitis and acute decubitis was very considerably higher than at the present day. Drozda (35) studied 927 cases in the three main hospitals of Vienna, and tells us that 43·12 per cent. recovered to such an extent as to leave the hospital; the fatal cases (56·68 of the whole) were all verified after death, but of course some of the patients that recovered may have been suffering from thrombosis.

The following table shows the relative mortality in the present series :—

	Number of cases.	Percentage dying within 24 hours.	Percentage dying within a week.	Percentage dying within a month.
Cerebral Hæmorrhage	109	37·7	72·5	86·2
Intraventricular Hæmorrhage	57	59·7	89·7	98·3
Extraventricular Hæmorrhage	52	15·4	53·8	73
Thrombosis	23	8·7	30·4	60·8
Embolism	13	0	30·7	30·7

It will be noticed that when blood flowed into the *ventricles*, nearly 60 per cent. of the patients died in the first twenty-four hours. One patient lived forty minutes, another thirty and one only fifteen, recalling the oft-quoted case of Abercrombie's (36) that proved fatal within five minutes of the initial symptom. These facts clearly negative such statements as Vlantassopoulo (37), who, endorsing a remark to the same effect by Lancereaux, says that the gravest hæmorrhage, even when accompanied by ventricular or meningeal inundation, never leads to death in a time shorter than some hours. Two of the patients in this series who lived for nearly a month, and another one who survived six weeks, presented *post-mortem* evidence that a clot had been present in their ventricles throughout.[1]

Of the *extraventricular* cases, the one running the most rapid course proved fatal in ten hours; the longest course was five years. Mushet (10) has published a case of this nature that died in thirty minutes, so that this rapid course is not confined to ventricular bleeding. On the other hand, cases living for twenty years are extremely rare, whereas with thrombosis they are quite common. Only three of Gintrac's 650 cases in which the duration of survival was stated, lived for ten years. I have compiled details from the published notes of *828 cases* in which cerebral hæmorrhage (whether intra- or extra- ventricular) was found after death, 650 having been collected by Gintrac (38), 43 by Charcot and Bouchard (39), 17 by Andral (40), 9 by Mushet (10), and 109 being of the present series. Of these 828 cases 30·4 per cent. died within twenty-four hours, 63·8 per cent. within a week, and 79·4 per cent. within a month. These percentages, although the material on which they are based is extensive, are probably rather too high, for the following reasons. They are obtained from hospitals, where the worst cases are taken; they are mostly published cases—and the special points for which they were published may often relate to circumstances of the onset, so that such a series would select against old-standing cases; and they are cases treated in the pre-aseptic era, when the mortality was unnecessarily high. On the other hand, Drozda's results, based on an even larger number, are to be much discounted by doubt as to the diagnosis of his recovering cases; in any case, his method must have caused selection of his data in favour of the apparent recovery from cerebral hæmorrhage. Summing up, therefore, it would seem probable that, even if the mild cases were included, quite half die within a week, and that a half of these die within the first twenty-four hours; whilst of the usual run of hospital cases the mortality is distinctly higher than this.

---

[1] *See* Case of Charcot's quoted above.

Taking now the subject of *thrombosis,* early death is certainly rarer here than with hæmorrhage. Nothnagel (41) says that there are only a few cases on record in which death took place within twelve hours; he refers here to both the occluding lesions. One of the present cases proved fatal in two hours; one, on the contrary, lived eighteen years. I have notes of *158 cases* of thrombosis, 106 being from Andral (40), 29 from Gintrac (42), and 23 being in the present series. The mortality of the whole was more rapid than in the present cases alone, while it will be remembered that in the case of hæmorrhage the mortality in the cases gathered from all sources was rather lower than in the present series alone. Of the 158 cases of thrombosis, 1·58 per cent. were fatal in the first twenty-four hours, 38 per cent. within the first week, and 74·7 per cent. in a month. It is striking that these figures, as far as they go, shew that hæmorrhage patients die twice as often as thrombosis ones in the first twenty-four hours, whereas in a month the deaths seem to be practically the same number in both instances. That this difference in the early deaths may really be even greater than here appears is suggested from the fact that Andral's cases showed an unusually fatal tendency. Thus only two out of his 106 patients lived for a year (43), which is certainly discordant from the experience of infirmary work, where such very large numbers of these older cases may be seen.

As regards *embolism*, the problem is complicated greatly by the commonly co-existing heart disease, and that death is determined more often by the latter lesion has been well pointed out by Newton Pitt (13) and Thevenet (44); the former observer states that only 40 per cent. of the deaths in his series of ninety-five embolisms were due to the cerebral lesion. His series, however, contained an unusually large number of cases of malignant endocarditis, in many of which the embolism was not even suspected until after death. The suggestion from Pitt's series is that the occurrence of cerebral embolism increases the seriousness of the outlook by indicating the probability that the infectivity of the endocardial lesion is reaching a higher degree of virulence and approximating to the malignant variety. The longest span of life in any of the present cases was eight months after the accident. As in the case of thrombosis, a considerably smaller number of cases of embolism than of hæmorrhage succumb in the first twenty-four hours; in the present series not a single case did so. I have collected notes of *273 cases* of cerebral embolism in which the length of survival was stated, 127 having been published by Eliza Walker (12), 55 by Newton Pitt (13), 34 by Gintrac (45), 8 by Jeannin (46), 36 by Lancereaux (26), and 13 in the present series. Of these only 8 per cent. died in the first twenty-four hours, 35 per cent. died within the first week, and 56 per cent. within the first month. Of Pitt's cases

21·8 per cent. died in the first twenty-four hours, whilst of all the other series only 4·5 per cent. did so.

Lesion.	Number of cases collected.	Percentage dying within 24 hours.	Percentage dying within a week.	Percentage dying within a month.
Hæmorrhage	828	30·4	63·8	79·4
Thrombosis	158	15·8	38·0	74·7
Embolism	273	8·0	35·5	56

(2) *Relation of Survival to Age and Sex.*

I have investigated only cerebral hæmorrhage in this connection, as the numbers are too few in the other lesions. The following tables refer, the first to the subject of age alone, the second to sex alone, and the third to sex at different ages.

I.

	Number of cases.	Percentage dying within 24 hours.	Percentage dying within a week.	Percentage dying within a month.	Age.
Present series	76	36·8	60·5	85·8	Under 60
Collected cases ..	350	32·8	59·0	77·0	
Total ..	426	33·3	59·3	77·9	
Present series ..	36	19·4	60·0	77·7	Over 60
Collected cases ..	327	26·6	61·8	79·2	
Total ..	363	25·8	61·4	79·0	

II.

	Number of cases.	Percentage dying within 24 hours.	Percentage dying within a week.	Percentage dying within a month.	Sex.
Present series	85	36·5	67	83·5	Male
Collected cases ..	392	31·4	58·9	75·3	
Total ..	477	32·2	60·3	76·7	
Present series	37	24·3	48·6	75·7	Female
Collected cases ..	285	27·7	62·4	82·1	
Total ..	322	27·3	60·8	81·3	

III.

	Number of cases.	Percentage dying within 24 hours.	Percentage dying within a week.	Percentage dying within a month.	Sex.
Under 60	259	35·9	59·8	76·8	Male
	167	29·9	58·6	79·6	Female
Over 60	210	26·6	60	75·7	Male
	153	24·8	63·4	82·4	Female

These figures are too few to be at all conclusive, but the results they point to, as far as they go, are these. From the first table we see that a higher percentage under 60 years of age die in the first twenty-four hours than of those over 60, but that this difference is more than abolished before the first week is out. According to the second table, it is the men who suffer most heavily in the first day, and the mortality in women does not exceed that in men until after the first week. The third table combines the other two, and from it we conclude that the younger men die on the first day in greater numbers than in any other class; the older women, who survive the first day best, are more prone than any other class to die in the next few weeks.

SITE OF THE LESION.

This is a subject which has been worked out fairly fully, at all events in the case of cerebral hæmorrhage, by Andral, Gintrac, Brouardel, Drozda, and other writers. For this reason, and also because the material in the present series does not comprise sufficiently precise information on many of the necessary points, I shall not attempt to deal with more than one of these points, namely, the side of the lesion. Some other details of the present series, however, will be given, on the chance of their proving of service for further studies.

(1) *Side of the Lesion.*

It has for a great many years been taught that hemiplegia due to cerebral hæmorrhage is most often a right-sided one, whilst that due to either an occluding lesion or hysteria is usually a left-sided one; this is often asserted to be of considerable value in diagnosis. It is interesting to observe that different writers have attempted to explain these differences by inventing quite contradictory anatomical data. Thus, Portal (47) says that cerebral hæmorrhage is more common on the right side because the right carotid is in a more direct line with the aortic stream than the left, so that the right side of the brain is more exposed to the force of the heart beats; while Rühle (48) says that cerebral embolism is more often met with on the left, because the left carotid is in a more direct line with the aortic stream than the right, so that fragments are swept to the left side of the brain rather than to the right. To account for the "paradoxical" cases of embolism on the right side, Jeannin (49) suggests that in these instances a left brachio-cephalic (innominate) artery is present, though he says that he has never been able to find a record of such an occurrence, and Cohn (50) suggests that in such cases the embolism has originated in the right innominate trunk or internal carotid artery. Without lingering any more on these ingenious hypotheses, however, let us examine the facts.

First, let us consider *cerebral hæmorrhage.* The dictum that this occurs more often on the right side has been handed down to us from Morgagni (51), who, basing his opinion on fifteen autopsies, came to the conclusion that the lesion was three times as common on the right side as on the left. Gendrin (52) went further when, from a study of forty-two autopsies on cerebral hæmorrhage cases, he concluded that this lesion was *five* times as common on the right side as on the left; this result is as startling as others of this author that we have referred to above. Larger numbers, however, tell a different story. Of 118 cases in the present series 68 showed hæmorrhage on the right side. I have collected a series of *995 cases* made up as follows: Gintrac (53), 373; Drozda (35), 282; Tooth (54), 76; Durand-Fardel (70), 117; Rochoux (56), 35; present cases, 118. Of this total number 496 showed hæmorrhage on the right side of the brain and 503 on the left. Under these circumstances the necessity of investigating the hydraulics of the aortic circulation in this connection becomes less pressing.

Turning now to the subject of *thrombosis*, we meet with a similar conflict of statements. Most authorities are agreed that the lesion is commonor on the left side, but whereas Leandier (57), writing in Bernheim's System, says that it occurs "almost always" on the left side, Andral (40) insists that it is rather commoner on the right side, and Dana (58) says that one side is as often affected as the other. In the present series of twenty-three cases, nine were right-sided and fourteen left-sided. Of a series of *176 cases*, in which the lesion was unilateral, Durand-Fardel (55) 140, Lancereaux (26) 11, Eliza Walker (12) 2, present series 23, there were eighty-three on the right side and ninety-three on the left.

It is with *embolism* that writers are most certain of the predilection of the lesion for one side. Nothnagel (59) says that it occurs "far more often" on the left side, and there are only a few authorities, such as Oppolzer (60) and Duroziez (61), who are not convinced of this preponderance, though the latter author is only sceptical on this point in cases in which the preceding heart disease is due to rheumatism. James Taylor (62) says that 60 per cent. of the lesions are left sided. Of the present cases five were on the right side and eight on the left. I have collected *558 cases* of unilateral lesions, from the following sources:—Saveliew (23) 65, Strauss (63) 22, Bertin (64) 38, Meissner (65) 32, Gintrac (45) 40, Eliza Walker (12) 117, Lancereaux (26) 39, Thevenet (66) 35, Duroziez (61) 68, Newton Pitt (13) 89, and present series 13. Only Saveliew's personal cases are counted, since his collection includes many of the cases elsewhere collected. Series such as Jeannin's (16) and Boé's (67), which were specially selected to show features in a way that would vitiate the enquiry now made, left hemiplegia being the thesis of the first work and aphasia of the second, have not been included. Of the total number 236 had right sided and 322 left, so that the percentage incidence of the latter was, as James Taylor surmised, practically 60. I know of no similar collection that contains more than a third of the numbers here given.

To summarise the above question, we may say that of 995 cases of hæmorrhage, 176 of thrombosis and 558 of embolism, a right-sided lesion was present in 49·89 per cent. of the first, 47·2 per cent. of the second, and 40·48 per cent. of the third. We have therefore no evidence in support of any predilection for one side on the part of the first two lesions, and even in the third case the evidence is not conclusive. It can be shown mathematically that when a given chance, *e.g.*, a right-sided lesion, appears 236 times in a series of 558, under such circumstances that only two options are open, *e.g.*, right and left (for all median and bilateral lesions were excluded), then all we are justified in concluding from such data is that, if a similar series were investigated in larger numbers, the chance would appear somewhere between 187 and 279 times per 558 trials; in other words, the true percentage would be, not necessarily 40·48 per cent., but some hitherto undetermined percentage, which would lie between 31·75 per cent. on the one hand and 49·11 per cent. on the other. So that, as far as we can say at present, embolism may be equally common on the two sides, or it may be twice as common on the left as on the right; and it is as likely to be the one as the other. This series does show, what no previous series that I am aware of has shown with certainty, that at all events embolism cannot be much commoner on the right side than on the left. We have further to bear in mind that most of the above series were collected by observers who were previously convinced of the partiality of embolism for the left side, and this may have affected the collecting process in some instances. It may well turn out, therefore, that embolism is in reality equally frequent on the two sides, or even more frequent on the right; it cannot be much more frequent on the right, and may turn out to be decidedly more so on the left. I do not think that we are in a position at present to make any more precise statement on the subject.

(2) *Position of the Lesion.*

Of the 123 cases of *Hæmorrhage* in the present series, only five were primarily pontine, though, of course, the pons was frequently implicated secondarily to hæmorrhage in the hemisphere. Primary pontine hæmorrhage occurred in nineteen of Tooth's 118 cases (54), in two out of 751 of Gintrac's cases (68). Cortical (non-meningeal) hæmorrhage was only present in two instances of the present series. Rupture of the hæmorrhage into the ventricles took place in fifty-two out of 109 cases of the present series in which the point was noted; this incidence, 47·7 per cent., is remarkably close to that observed by Tooth (69) in his 118 cases, where it was 47·4 per cent., but not to that recorded by Gintrac (68), which was only 6·1 per cent. of 751 cases. In the present series, blood was found in both lateral ventricles twice as often as in one alone. When found in both lateral ventricles, it was present in the third and fourth ventricles in eight-ninths of the cases. Of the eighteen cases showing blood in the fourth ventricle as well as the lateral, in only two was it present in one lateral ventricle only.

As regards *occluding lesions*, the middle cerebral artery was naturally the one usually affected, for the series was selected by the existence of hemiplegia. The basilar alone was once thrombosed, as was the artery of the crus cerebri. Thrombosis affected the cortex alone in six instances, the internal capsule alone in five, and both in eight. One, three and eight were the corresponding numbers with embolism.

CONCLUSIONS.

The most definite conclusion to which these investigations point is that we stand in great need of a large series of cases of vascular lesions, the history of which has been critically studied, and the evidences of the lesion before and after death carefully recorded. The following are the results of the present inquiry that seem to rank highest in the grade of probability, but until we have better evidence before us, they are to be looked upon as purely tentative efforts to reach the truth.

(1) Rest in bed, and especially sleep, protect to some extent against cerebral hæmorrhage. This would seem a more accurate statement than that rest and sleep predispose to thrombosis, as I can find no evidence in favour of this,

though in actual practice the two statements have a similar bearing on diagnosis.

(2) Severe exertion and time of day appear to have had too much stress laid on them in the past. A miliary aneurism that is going to rupture rarely needs the aid of severe exertion for this consummation. Time of day is only of interest when the habits of the patient's blood-pressure at different hours are known.

(3 Consciousness is lost at the onset in half the cases of occluding lesions and three-quarters of hæmorrhage lesions. It is transitorily affected in many of the remaining cases, but we have no accurate knowledge as to the depth or the duration of the coma in the various lesions, its relation to the intensity of the paralysis, or many other important questions. Until these latter are decided, it is well to remember the danger of relying too greatly on this aid to diagnosis, on account of an important group of occluding lesions in which the onset is apoplectiform, and another group of hæmorrhage lesions in which the onset is quite insidious.

(4) The immediate prognosis is much graver when the onset is apoplectiform; especially is this so in cases of hæmorrhage. Contrariwise, cases of late hemiplegia due to hæmorrhage are less likely to have suffered from loss of consciousness at the onset than hæmorrhage cases taken as a whole, and are therefore the more likely to be attributed to thrombosis.

(5) Intraventricular hæmorrhage, which is nearly always secondary, may not cause loss of consciousness. On the other hand, immediate loss of consciousness as the initial symptom may be due to extraventricular hæmorrhage.

(6) The immediate prognosis is much graver in cases of hæmorrhage than in cases of occluding lesions. In 828 cases of hæmorrhage, 158 of thrombosis, and 273 of embolism, the results are as follows: Over 30 per cent. of the cases of the first lesions were fatal within twenty-four hours, half as many of the second lesion, and a quarter as many of the last lesion; almost two-thirds of the first patients are dead in a week, and more than a third of the patients with occluding

lesions. Of twenty cases of each lesion four hæmorrhage ones would survive a month, five thrombosis ones and nine embolism ones. Most of those that survive two years are cases of thrombosis.

(7) Of the cases in which blood is found in the ventricles, 60 per cent. die in the first twenty-four hours and 90 per cent. in the first week. It is not very rare, however, for such cases to live a few weeks.

(8) The mortality incidence is heaviest on the first day amongst the younger men; in the next few weeks it is the aged women who are most likely to die.

(9) There is no indication that hæmorrhage affects the right side of the brain more often than the left. Occluding lesions may affect the left side more than the right for all we know to the contrary, but there is no evidence that is at all conclusive in favour of this supposition, and the statements commonly made on the subject are not warranted in the present state of our knowledge.

In conclusion, it is my pleasant task to offer my sincere thanks to Sir William Gowers for the kindness he has shown me in connection with this contribution; I am also indebted to the Committee of University College Hospital for permission to make use of their records.

REFERENCES.

(1) GINTRAC. "Maladies de l'Appareil Nerveux," 1869, T. ii., pp. 440, 441.
(2) DANA. "Twentieth Century Practice of Medicine," vol. x., p. 292. "Dercum's Text-Book of Nervous Diseases," 1896, p. 455.
(3) CHARCOT. "Notes Inédites." Quoted by Brouardel in *Dict. Encycl. des Sc. Méd.*, 1873. 1st. S., T. xiv., p. 371.
(4) SORMANI. Quoted by Brouardel, *Op. cit.*, p. 359.
(5) VLANTASSOPOULO. "De l'Hérédité dans l'Hémorrhagie Cérébrale." *Thèse de Paris*, 1894, p. 71.
(6) THEVENET. "Contribution à l'étude du diagnostic différentiel entre l'hæmorrhagie et l'embolie cérébrale." *Thèse de Lyon*, 1894, p. 26.
(7) GENDRIN. "Traité Philosophique de Médec. Pract.," 1838, vol. i., p. 554.
(8) TOOTH. "Allbutt's System of Medicine," 1899, vol. vii., p. 608.
(9) JAMES TAYLOR. "Allbutt's System of Medicine," 1899, vol. vii., p. 563.
(10) W. B. MUSHET. "Practical Treatise on Apoplexy," 1866, p. 21, *et seq.*
(11) W. BROADBENT. *Med. Chir. Review*, 1887, p. 335.
(12) ELIZA WALKER. "Ueber die Verstopfung der Hirnarterien." *Inaug. dissertation der Universtät, Zurich*, 1872.

(13) Newton Pitt. "Goulstonian Lectures," 1890. *Brit. Med. Journ.*, April 12, 1890, p. 829.
(14) Thomas Jones. *Brit. Med. Journ.*, 1864, vol. ii., p. 658.
(15) Trousseau. *Gazette des Hôpitaux*, Feb. 2, 1841.
(16) Jeannin. "Contrib. à l'étude de l'embolisme cérébrale de la Sylvienne droite." *Thèse de Paris*, 1894, p. 52.
(17) Leandier. Bernheim; "Traité Pratique de Médecine," 1897, T. ii., p. 67.
(18) Vlantassopoulo. *Op. cit.*, p. 70.
(19) Trousseau. Clinique Méd. de l'Hôtel Dieu, T. ii., p. 57.
(20) Lancereaux. "De la Thrombose et de l'Embolie cérébrales. Paris, 1862, p. 60.
(21) Nothnagel, in "Von Ziemmsen's Cyclopædia of Medicine," English Transl., 1877, vol. xii., p. 195.
(22) Monakow. "Nothnagel's Specielle Pathologie und Therapie," 1897, Bd. ix., Alt. 1, S. 864.
(23) Nicolas Saveliew. *Virchow's Arch. fur Path. Anat. und Phys. i. f. Klin. Med.*, Berlin, Jan. 10, 1894, Bd. cxxxv., Hft. 1, S. 112, *et seq.*
(24) Gintrac. *Op. cit.*, p. 447.
(25) Thevenet. *Op. cit.*, p. 28.
(26) Lancereaux. *Op. cit.*, Appendix.
(27) Gintrac. Quoted by Brouardel, *loc. cit.*, p. 371.
(28) Charcot. "Clinical Lectures on Senile and Chronic Diseases." *Trans. of New Sydenham Soc.*, by W. S. Tuke, 1881, N.S., vol. xcv., p. 272.
(29) Durand-Fardel. "Traité de Ramollissement du cerveau," 1843, p. 126.
(30) Tooth. *Loc. cit.*, p. 626.
(31) Copland. "Dict. of Pract. Medicine," vol. i., p. 82. See also "On the Cause, Nature and Treatment of Palsy and Apoplexy," London, 1850.
(32) Brissaud. Bouchard and Brissaud's "Traité de Médecine," 1904, T. ix., p. 213.
(33) Brissaud. *Loc. cit.*, p. 216.
(34) Dana. "Twentieth Century Practice of Medicine," vol. x., p. 278.
(35) Drozda. "Statist. Stud. uber die Haem. cerebr." *Wien. Med. Presse.* 1880, Jahrgang xxi., S. 299, *et seq.*
(36) Abercrombie. "Researches on Diseases of the Brain and Spinal Cord."
(37) Vlantassopoulo. *Op. cit.*, p 66.
(38) Gintrac. *Op. cit.*, p. 481.
(39) Charcot and Bouchard. *Arch. de Phys.*, 1868, T. i., p. 112, *et seq.*
(40) Andral. *Clinique Médicale*, 1833, T. v.
(41) Nothnagel. *Loc. cit.*, p. 20[illegible].
(42) Gintrac. *Op. cit.*, vol. i., p. 446.
(43) Andral. *Op. cit.*, p. 588.
(44) Trevenet. *Op. cit.*, p. 41.
(45) Gintrac. *Op. cit.*, vol. i., p. 468.
(46) Jeannin. *Op. cit.*, p. 18, &c
(47) Portal. "Traité de l'Apoplexie," p. 333.
(48) Rühle. Quoted by Jeannin. *Op. cit.*, p. 9.
(49) Jeannin. *Op. cit.*, p. 9.
(50) Cohn. "Klinik der embolischen Gefässkrankheiten," Berlin, 1860.
(51) Morgagni. "De Sedibus et Causis Morborum." Quoted by Durand-Fardel. *Op. cit.*, p. 309.
(52) Gendrin. *Op. cit.*, p. 517.

(53) Gintrac. *Op. cit.*, T. ii., p. 468.
(54) Tooth. *Loc. cit.*, p. 615.
(55) Durand-Fardel. *Op. cit.*, p. 309, *et seq.*
(56) Rochoux. "Recherches sur l'Apoplexie," Paris, 1814, p. 195.
(57) Leandier. *Loc. cit.*, p. 72.
(58) Dana. "Twentieth Century Practice of Medicine," vol. x., p. 269.
(59) Nothnagel. *Loc. cit.*, p. 184.
(60) Oppolzer. *Wien. Méd. Woch.*, 1859, S. 50, and 1860, S. 1.
(61) Duroziez. "Du Rétrécissement mitral pur," Paris.
(62) James Taylor. *Loc. cit.*, p. 562.
(63) H. Strauss. *Inaug Disser.*, Berlin, 1896.
(64) Bertin. *Schmidt's Jahresberich.*, Bd. cxlvii., S. 288. "Etude critique de l'embolie," 1869.
(65) H. Meissner. *Schmidt's Jahresber.*, 1861, T. cix., p. 89.
(66) Thevenet. *Op. cit.*, p. 95, *et seq.*
(67) Boé. "Aphasies dans les maladies du cœur." *Thèse de Paris*, 1880.
(68) Gintrac. *Op. cit.*, T. ii., p. 482.
(69) Tooth. *Loc. cit.*, p. 616.
(70) Durand-Fardel. Traité Pratique des Maladies des Vieillards," Paris, 2^{me} ed., 1873, p. 185.

Reviews.

SOME RECENT RESEARCHES ON THE TRACTS OF THE BRAIN-STEM.

(1) *Untersuchungen über die Leitungbahnen des Truncus cerebi.* M. LEWANDOWSKY. *Neurobiologische Arbeiten.* (O. Vogt.) Erster Band; Zweite Lieferung.

(2) *Experimentale und Pathologisch-anatomische Untersuchungen über den Verlauf einiger Bahnen des Centralnervensystems.* S. MINGAZZINI. *Monatschrift für Psychiatrie und Neurologie.* Bd. xv., 1904.

(3) *Les Pédoncules cérébelleux supérieurs.* A. VAN GEHUCHTEN. *Névraxe*, vol. vii., 1905.

(4) *Le Corps Restiforme et les connexions bulbocérébelleuses.* A. VAN GEHUCHTEN. *Névraxe*, vol. vi., 1904.

THESE four papers may be grouped together for the purpose of a review of recent work on the anatomy of the brain-stem, as they all deal with structures of this region, though individual parts naturally receive more attention from the one or other author. Reference is occasionally made to other publications which tend to substantiate or controvert the conclusions of the authors under discussion.

Lewandowsky's monograph deserves close attention. This is, in the first place, due to the large amount of material on which his conclusions are based, the study of complete series of forty brains which had been subjected to experimental lesions—dogs and cats were the subjects of his experiments; and secondly, to the application of the method of Marchi, which is now almost universally admitted to be that most capable of giving positive and definite results when employed with reasonable care. If anything can be urged in adverse criticism of the work, it is that the experimental lesions appear to have been so extensive that in many instances the isolation of tracts could have been obtained only with difficulty and at the risk of confusion.

Mingazzini's work, on the other hand, results from the study of the central nervous system by Weigert's stain. His material consisted of five human brains in which destructive processes had developed during life, and of the brains of three animals on which Luciani had operated. The method he has used is of course not capable of yielding such positive results as that which is known by the name of Marchi, but in experienced hands the careful observation of the secondary and tertiary degenerations which follow local lesions may give much information on the origin and coupling up of the systems of fibres concerned.

van Gehuchten's papers bear the imprint of the careful work of their author. They result from the study of degenerations from experimental lesions in the brains of rabbits by Marchi's method.

Cerebellum.—The recent work of Clarke and Horsley (1) leads to the conclusion that none of the efferent cerebellar fibres take origin in cerebellar cortex, and that the axones of these cells only reach the central nuclei. van Gehuchten's results entirely confirm these observations, but he has not attempted, as these authors have, to determine the more precise relations between the two structures. According to Mingazzini, on the other hand, the cortex of each lateral lobe sends fibres to the opposite nucleus dentatus.

Superior Cerebellar Peduncles.—According to van Gehuchten the fibres of the superior peduncles arise from the cells of the nucleus dentatus, and probably also from those of the roof nuclei. Lewandowsky inclines to the same opinion, but has not been able to bring its definite proof. Both authors are agreed that all the fibres of this peduncle are cerebellofugal, but Mingazzini describes a considerable proportion of them as cerebellopetal. According to van Gehuchten none of the peduncular fibres decussate in the vermis, but Lewandowsky recognises such fibres, and names them the tractus uncinatus (identical with the faisceau en crochet of Russell and Thomas). The fibres of this tract arise (according to the investigations of Probst) from the cells of the nucleus tecti, and leave the cerebellum in its superior peduncle lying in its dorsal part during its course through the mid-brain. Here the tract bifurcates, the one branch, fasciculus retropeduncularis, ends around cells between the peduncle and the fillet, the other (fibræ cerebellovestibulares) passes caudalwards along the inner side of the corpus restiforme, and ends in the nucleus dorsalis acustici (? nucleus of Bechterew), and in the descending vestibular root.

The authors who have worked with Marchi's methods are in full agreement on the extracerebellar course of the superior peduncle. Its decussation in the midbrain is complete, none of its fibres pass further oralwards than the decussation on the homolateral side. After decussation the tract breaks up into two branches, an ascending and a descending. The descending branch bends caudalwards through the pons, gradually diminishing in bulk and becoming more median in position. Its fibres seem to end in the pontine gray matter, chiefly in the nucleus reticularis tegmenti (of Bechterew), but some could be followed almost to the inferior olives (Lewandowsky). To van Gehuchten, however, it seemed that the exact termination of these fibres could not be determined. The larger ascending branch gives off fibres and collaterals to the nucleus ruber, but its chief mass enters the lamina interna thalami, and ends, according to van Gehuchten in the median portion of the thalamus, according to Lewandowsky in the nuclei ventralis a., medialis b., and nucleus lateralis anterior (of Monakow). Neither author was able to trace fibres to the other basal ganglia, to the internal capsule, or to the cerebral cortex. van Gehuchten describes a second decussation of some of the fibres of the superior peduncle in the thalamus, the one branch passing ventral to the third ventricle into the opposite thalamus, *i.e.*, that homolateral to the lesion.

Lewandowsky believes that the superior peduncles are not joined by any fibres from the nucleus ruber during their oralward course.

Neither van Gehuchten nor Lewandowsky has been able to trace a tract leaving the cerebellum by its superior peduncles to pass to the spinal cord (descending cerebellar tract of Cajal), and both have been unable to find any evidence of the neutral cerebello-thalamic tract described by Probst (5). According to Probst's first description this tract leaves the cerebellum in its superior peduncles, then turns medialwards in the tegmentum of the pons and decussates with its fellow in the raphé; it then runs oralwards dorsal to the fillet and through the capsule of the nucleus ruber to reach the ventral nucleus of the thalamus. In later publications (3 and 4) Probst described additions to this tract from the region of the spinal root of the nervus trigeminus and from Deiters' nucleus.

According to Mingazzini's views the constitution of the superior peduncles is more complicated. He admits that the majority of its fibres are cerebellofugal, but these end round the cells of the caudal end of the nucleus ruber, and from there a second link of neurones connects the system with the ventral portion of the thalamus, and possibly a third set of fibres passes from here to the cerebral cortex. He does not recognise a descending branch of the peduncle. It also contains cerebellopetal tracts. Fibres from the cerebral cortex (from the frontal and possibly too from the temporal lobe) reach a not yet defined centre in the tegmentum which must lie oral to the nucleus ruber, and from it a second set of neurones arises which decussate with the cerebellofugal fibres, and pass to the cerebellum through its superior peduncles. The capsule of the nucleus ruber is formed by superior peduncular fibres, and fibres which arise in this nucleus join the peduncles during their decussation. Mingazzini's conclusions also tend to confirm the existence of a decussation of peduncular fibres in the cerebellum.

Sand (8) also describes cerebellopetal fibres in the superior peduncles in man as a result of examination of material by Marchi's method. According to him, however, they come from the cortex or the corpus striatum, or from both.

Middle Cerebellar Peduncles.—The information on the middle cerebellar peduncle is less definite. Lewandowsky concludes that it contains chiefly cerebellopetal fibres from the contralateral pontine nuclei, and possibly, but in smaller number, also cerebellofugal fibres to the pons, as Thomas has described. One such tract runs within the median part of the peduncle to the nucleus centralis superior internus.

Mingazzini considers the system in relation to the corticopontine tracts and the fibræ transversales of the pons. Cerebellofugal fibres reach the homo- and contra- lateral sides of the pons, and from here other neurones pass oralwards with the contralateral pyramidal tracts through the middle fifth of the pes pedunculi. Other cerebellofugal fibres end in the nucleus reticularis tegmenti. The descending systems consist of fibres from the frontal lobe which end in the dorsomedian cells of the pons, whence other axis cylinders spring, which pass through the strata profundum et superficiale into the opposite middle peduncle; and fibres from the temporal lobes which pass through the lateral fifth of the pes to the lateral pontine nuclei and to the nucleus reticularis; and from here a second link passes to the opposite peduncle; finally, the pyramidal tracts give fibres or collaterals to the cells of the stratum complexum pontis.

According to Lewandowsky the fibræ olivo-cerebellares are the chief constituent of the corpus restiforme. All these are afferent to the cerebellum, *i.e.*, the cerebellum does not emit any fibres to the inferior olives. As the majority of these fibres decussate in the medulla this connection of the olives with the cerebellum is contralateral. They end in the lateral lobe, in the flocculus, and possibly too in the vermis. The tractus spino-cerebellaris dorsalis (Flechsig's tract) is also an important constituent of the inferior peduncles; it terminates in the anterior part of the vermis. The dorsal columns of the spinal cord do not send fibres directly to the cerebellum, nor do any fibres of the dorsal column nuclei reach it. Each corpus restiforme contains two descending tracts. (1) Fibres which pass down with it course round the same side of the medulla as fibræ arcuatæ externæ ventrales, and bend dorsalwards round the pyramidal tract of the same side, to end in the nucleus raphé, a small collection of cells which lies between and dorsal to the pyramids. (2) The other tract consists of the fibres to Deiters' nucleus. van Gehuchten, whose results were obtained by experiments on rabbits, concludes that the corpora restiformia do not contain any cerebellofugal fibres. Besides the direct cerebellar tract and the fibræ olivo-cerebellares they contain fibres which take origin in the bulb; of these the most important are those which arise in connection with the cells of the nucleus lateralis medullæ, and end chiefly in the homolateral side of the vermis. Other fibres take origin in the reticular formation of the medulla, the bundles can be traced from its ventral and dorsal portions—fibræ reticulo-cerebellares ventrales et dorsales. Their relation to the cerebellum is chiefly direct, partly crossed. His results agree with those of Lewandowsky to the exclusion of spinal fibres and of fibres from the dorsal column nuclei in the corpus restiforme.

Mingazzini describes two cerebellofugal systems in the inferior peduncles; the one bundle passes to the opposite inferior olive, and the other consists of fibres which decussate in the ventral portion of the raphé, and form the fibræ peripyramidales of the opposite side. They end in the nucleus arciformis. He also draws attention to the participation by the accessory olives in the distribution of the cerebellofugal fibres.

Secondary Motor Tracts.—The chief efferent spinal tracts of the cerebellar system, described by Lewandowsky as those from Deiters' nucleus, and from the nucleus ruber. The fibres from Deiters' nucleus are, after their origin, diffusely scattered through the cross section of the medulla, and first collect into a definite tract, dorsolateral to the inferior olives. In the ventrolateral columns of the upper cervical segments of the spinal cord they are still scattered, and almost surround the ventral horn in which they end; the majority are in homolateral relation with the cord, but a small proportion decussate in the pons a short distance from their origin. As the group of cells known as Deiters' nucleus has no connection with the vestibular nerve, this tract should not receive the name vestibulospinal. Nothing new is added as to the course of Monakow's bundle. It rises from the cells in all parts of the nucleus ruber, and not from only those of an isolated region as Kohnstamm and many other observers describe. It certainly receives no fibres from the thalamus or the neighbouring gray matter. Further, it contains, as far as the available evidence goes, the only efferent fibres given off by the nucleus ruber. Preisig (2), however, from examination of retrograde tigrolysis by Nissl's method, comes to the conclusion that its anterior cells send axones oralwards, and that only the cells of its caudal half supply fibres to Monakow's bundle. After its decussation and during its course through the pons Monakow's bundle is joined by fibres from the contralateral pontine nuclei (fibræ pontospinales). Some of its fibres end in, or give collaterals to, the nucleus lateralis medullæ, but the majority enter the lateral columns of the spinal cord, and probably end in the intermediate gray matter.

The tractus tectospinalis springs from the large cells of the stratum griseoalbum medium of the anterior quadrigeminal bodies. It takes the course which has been already fully described. In the pons and bulb it gives off many fibres, especially in the direction of the facial nuclei, though it is doubtful if any of its fibres actually have this destination. Further caudalwards it fuses with the dorsal longitudinal bundle, and passing through the ventral column of the cords splits up in the ventral horns.

The dorsal longitudinal bundle is a complex system of fibres with various origins and terminations. The descending tracts are: (1) Fibres which spring from the cells of Darkschewitsh's nucleus (nucleus of posterior commissure) and run in the median part of the bundle to the level of Deiters' nucleus, below which they lie ventral to it. They enter the ventral column of the spinal cord on the same side, without having any relation to the intermediate cranial nerve nuclei. (2) The tractus pontis descendens (Probst) which rises from the large cells in the neighbourhood of the motor trigeminal root. (3) Fibres from Deiters' nucleus. The latter two systems also end in the ventrolateral columns of the spinal cord. The chief ascending tract of the dorsal longitudinal bundle consists of fibres which spring from Deiters' nucleus and terminate in the third and fourth nerve nuclei. Probably they also provide ramifications to the abducens nucleus. In spite of previous assertions to the contrary the dorsal longitudinal bundle does not supply any root fibres to the oculomotor nerves.

Another tract which Lewandowsky mentions as ending in the third nerve nucleus is the tractus pontis ascendens described for the first time by him. It springs from the nucleus reticularis pontis or from the cells around it, decussates caudal to the decussatio brachii, and runs oralwards dorsal to the median fillet to the nucleus ruber, at the caudal end of which its fibres bend dorso-medialwards to enter the oculomotor nerve nucleus.

Hitherto, little has been known of the origin or destination of the group of fibres which in cross-section of the midbrain are known as Forel's bundle. Lewandowsky believes they have an extensive origin in the pons and medulla, some rise as far caudalwards as the inferior olives. The tract is represented by scattered fibres in the formatis reticularis medullæ, which collect into a more compact bundle ventral to the nucleus dorsalis acustici. Oralwards it takes the well-recognised course, and increases in size. It ends in the ventral portion of nucleus ventralis b. of the thalamus.

Sensory Systems.—The median fillet is another tract whose constitution has led to much discussion. From the work of Lewandowsky it would appear that it certainly does not contain any spinal fibres. It will be remembered that many text-books describe fibres from the ventro-lateral columns of the cord as one of its chief components. Probst, however, in a recent work confirms the existence of fibres of spinal origin in the fillet in man (6). Its chief source is from the dorsal column nuclei, by way of the fibræ arcuatæ internæ, all of which that enter the fillet decussate. The arrangement is such that the fibres from the nucleus gracilis go to form its lateral part, which thus contains the second link of the lumbosacral dorsal roots, while those from the nucleus of Burdach's column pass oralwards in its median portion. In the pons it receives fibres from the contralateral sensory nucleus of the trigeminus, but not from the substantia gelatinosa of the spinal root. The fillet does not give off fibres or collaterals till it reaches the level of the corpus geniculatum internum; there some of its fibres split up in a small collection of cells between this ganglion and the brachium posterius—nucleus parageniculatus. None of its fibres enter the lateral fillet or pass to either of the quadrigeminal bodies. All its fibres end in the thalamus, none pass to the basal ganglia, to the internal capsule, or to the cortex. The majority pass through the lamina medullaris externa, to end in nuclei ventralis and nucleus ventralis anterior; a smaller number enter the lamina medullaris interna and reach nucleus medianus. Lewandôwsky has not been able to find any evidence of the existence of the central sensory trigeminal tract which Wallenberg and others have described springing from the spinal trigeminal root, and he believes the fillet alone serves to conduct centralwards the sensory impressions from the face. These conclusions practically agree with those of Probst and Tschermak.

Wallenberg (9) has recently described two newly known tracts in connection with the main sensory nucleus of the trigeminus. The one consists of coarse fibres which end in motor trigeminal nuclei, in both central trigeminal nuclei and in the third nerve nuclei, but a few reach the thalamus. The other, which contains finer fibres, joins the contralateral fillet, and can be followed in it to the centre median of the thalamus.

Mingazzini concludes that the "chief fillet" contains both centripetal and centrifugal tracts. In the paper under consideration he only deals with the latter, but his arguments are inconclusive and his statements indefinite. His opinion seems to be that the chief origin of these centrifugal fibres is in the ventral nucleus of the thalamus, and their termination in the interolivary layer and the contralateral dorsal column nuclei. The so-called accessory bundles of the fillet are discussed in more detail, especially the fasciculus pontinolateralis. In the midbrain it lies ventral to the chief fillet, further caudalwards occupies its median portion, and at the level of the seventh nucleus the fibres begin to disappear, though some reach to the interolivary layer. He concludes that it springs from the cortex, and is consequently centrifugal, though it may contain centripetal fibres as well. He has not been able to decide finally if it serves in the conduction of cortical impulses to the cranial nerve nuclei, as Hoche and Theile have described.

To Lewandowsky it seems probable that all fibres which connect the pallium and thalamus are corticopetal, at least they have their cells of origin in the thalamus. The fibres which have been described as connecting the thalamus with the nucleus ruber do not exist, nor does the thalamus send any fibres to the spinal cord.

Auditory System.—The roots of the auditory nerve and their central connections have been carefully studied by Lewandowsky. The lateral (cochlear) root ends in the nucleus ventralis and tuberculum acusticum, from which three central tracts spring, viz., the corpus trapezoides; the fibræ acusticæ of Held, which bend round the dorsal edge of the corpus restiforme, and then ventralwards through the spinal trigeminal root, to reach the homolateral olive; and the striæ acusticæ of Monakow, which decussate in the raphé, and only join the corpus trapezoides in the opposite olive. These systems form the corpus trapezoides, which enters into relation with the contralateral superior olive and nucleus trapezoides, and to a less extent with those of the same side reinforced by fibres from these nuclei the corpus trapezoides forms the lateral fillet, and it has no other connections. After giving fibres to and receiving such from its nucleus, the lateral fillet ends in the posterior quadrigeminal bodies. The commissure, which Probst has described connecting the two nuclei of the lateral fillet, is easily demonstrated. It crosses the raphé just caudal to the decussatio brachii. These are the sole terminations of the lateral fillet; none of its fibres reach Deiters' nucleus, and none pass further oralwards than the posterior quadrigeminal bodies. The next link in the central cochlear system is brachium posterius, which ends in the internal geniculate body, though according to Mingazzini, there is a direct connection between the posterior quadrigeminal bodies and the temporal lobes. The lateral root of the eighth nerve does not, however, consist exclusively of cochlear fibres. In the first place, some of its fibres pass oralwards to form a tract lying in the lateral angle of the fourth ventricle median to the corpus restiforme, which ends in Bechterew's nucleus. The name fasciculus solitarius acustici is proposed for this. A second set of fibres of the lateral root bend caudalwards to enter the descending vestibular root.

The majority of the fibres of the median (vestibular) root bend caudalwards into the descending root, others end in the nucleus dorsalis acustici, chiefly in the homolateral, partly in that of the opposite side. No direct root fibres enter the cerebellum, there is no evidence to support the existence of Edinger's "directe sensorische Kleinhirnbahn."

The nature of the cerebral root of the trigeminus still remains doubtful. Certain only is it that the axis cylinders of its large cells descend and join the motor root, but it is not yet finally settled if these are the only fibres in contains, or if ascending fibres run in it.

Another bundle of fibres, which takes origin in the region of the cerebral trigeminal root, was first described by Probst, and to it Lewandowsky suggests that this worker's name should be applied. Probst's tract runs at first with the trigeminal rôot, and, after the exit of this, lies in the dorsolateral part of the formatio reticularis of the pons, in the neighbourhood of Deiters' nucleus. It ends in the gray matter, ventral to the nucleus dorsalis vagi.

The central tegmental tract certainly arises from the cells of the anterior quadrigeminal bodies, but it probably also receives fibres from the pontine nuclei in its caudalward course. It ends in the inferior olive. Probst (6), who failed to see this tract in animals, describes its origin in man from the nucleus ruber or its neighbourhood, but on unsatisfactory grounds.

The pyramidal system and cerebral peduncles contain no centripetal fibres. The greater bulk of the fibres end in the pontine nuclei, and the substantia nigra receives a considerable number. The pyramidal system has no relation to the fillet, it neither gives to or receives fibres from it, and Lewandowsky has been unable to find any evidence of the "Bündel zur Haube," or of the other accessory fillet systems which have been frequently described.

Sand (8) has come to the same conclusion from examination of human material by Marchi's method, though many fibres pass obliquely through the fillet on their way to the motor nerve nuclei. These discrepancies may perhaps be explained by Probst's observation that pyramidal fibres run in the fillet only in some brains. He describes the "Fussehleife" which lies dorsal to the pyramidal tract in the pes pedunculi, and the "Bündel vom Fuss zur Scheife," which leaves the pyramidal tract in the pes, winds round the medial border of the latter and passes dorsalwards into the fillet.

Lewandowsky has not been able to follow degenerated pyramidal fibres to the nuclei of any of the cranial nerves, though, at the level of the facial and hypoglossal nuclei fibres leave the pyramidal tracts and cross the raphé to the contralateral side. Similarly, degeneration can never be traced into the ventral horns of the cord; the myelinated fibres end in the intermediate gray matter, and they certainly do not enter Clarke's column, as Rothman (7) has suggested. Proximal to their decussation a few pyramidal fibres enter the homolateral olive, but none could be traced to the contralateral, as Probst and Rothman have described.

The tract of fibres in the lateral columns of the upper cervical cord described by Thomas and known by his name appears to Lewandowsky to take origin in the formatio reticularis of the caudal part of the medulla. Practically all of them end in the gray matter of the cervical cord, and they thus compose a conduction system between the medulla and the latter.

The exact origin of the two spino-cerebellar tracts is uncertain. According to Lewandowsky they are homologous systems, Gowers' tract carrying fibres from the sacrolumbar, the direct cerebellar tract from the dorsocervical cord. Both spring from the cells of Clarke's column or its representatives in the lumbosacral cord, and decussate almost immediately after origin. They give fibres to the nucleus lateralis medullæ as they pass it, and enter the cerebellum, ending chiefly in its homolateral half after passing through their well recognised course. The direct cerebellar tract ends in the anterior vermis, Gowers' in the posterior vermis. No fibres leave Gowers' tract to pass further oralwards than the cerebellum. Lewandowsky failed to find any spinal fibres ascending to the thalamus or to the midbrain.

Mingazzini, in a case of microcephaly, found a considerable number of fibres in the direct cerebellar tract, though no cells were visible in Clarke's column. He therefore concludes that this tract receives fibres from other cells, and probably, too, direct dorsal root fibres, an assumption entirely at variance with all accurate observations on the subject.

GORDON HOLMES.

(1) CLARKE AND HORSLEY. BRAIN, 1905, vol. xxviii., part 109, p. 13.
(2) PREISIG. *Journal für Psychiatrie und Neurologie.* Bd. iii.
(3) PROBST. *Archiv. für Psychiatrie.* Bd. xxxv.
(4) ,, ,, ,, ,, Bd. xxxiii.
(5) ,, *Monatschrift für Psychiatrie und Neurologie.* Bd. x.
(6) ,, *Sitzungsber. d. Kaiserl. Akadamie der Wissenschaft in Wien.* Bd. cxii., 3 Abth.
(7) ROTHMAN. *Archiv. für Anatomie und Physiologie.* Supplementband, 1903.
(8) SAND. *Arbeiten aus dem Neurolog. Institut in Wien.* Bd. x.
(9) WALLENBERG. *Anatomischer Anzeiger.* Bd. xxvi.

THE TICS.

(1) *Tics.* HENRY MEIGE. (*Monographies Cliniques sur les Questions Nouvelles.*) Masson et Cie., Paris, 1905.

(2) *Étude Critique sur le Tic Convulsif et son Traitement Gymnastique.* CRUCHET. Thèse de Bordeaux, 1902.

(3) *Bemerkungen zur Lehre vom Tic.* OPPENHEIM. *Journ. f. Psychologie u. Neurologie,* Bd. 1, 1902.

(4) *Discussion.* MEIGE, &c. *Congrès des Médecins Aliénistes et Neurologistes de France.* Limoges, 1901.

Discussion. MEIGE, PITRES, CRUCHET, GRASSET, &c. Grenoble, 1902.

Discussion. MEIGE, RUDLER, CHOMEL, DUPRÉ, &c. Bruxelles, 1903.

Discussion. MEIGE, FEINDEL, CRUCHET, RUDLER, CHOMEL, &c. Pau, 1904.

IT is unfortunate that our acquaintance with the word "tic" is to a great extent limited to the term "tic douleureux," for our conception of its meaning is unconsciously coloured by our interpretation of the phenomena of that condition. Now, whatever "tic douleureux" be, in the light of present-day knowledge it is assuredly no tic. Nor can we consider it satisfactory that the cognate phrase "habit spasm" should be in vogue, especially in this country, to express those reputedly harmless "nervous movements" with which we are all familiar, for it is no less inaccurate than it is misleading, and, moreover, it is gratuitously exclusive. A tic may arise from a habit as well as from other sources, but it itself is more than a habit; on the other hand, its earliest manifestation may be a spasmodic motor reaction, but it is certainly not a spasm. To the French school of neurology more particularly is due the credit of having made an honest attempt to extricate the word tic from its uncongenial surroundings, and to restore it to its proper estate. The fact that it is found in many languages would seem to show that it has a certain specificity of meaning, while perhaps its chief recommendation is that it is noncommittal.

In spite of recent advances, it is curious how indefinite medical science is in the handling of common terms such as spasm, convulsion, clonus, tremor, and it is pathetic to observe the refuge sought in the adoption of generalisations such as myotonus, myoclonus, myotetanus, myokymia, microkinesis, hyperkinesis, &c., for they only serve to obscure the issue. Uncertainty is so frequently attributable to a confused terminology that it is desirable Meige's latest definition should be quoted in full.

"A tic is a co-ordinated purposive act, provoked in the first instance by some external cause or by an idea; repetition leads to its becoming habitual and finally to its involuntary reproduction without cause and for no purpose, at the same time as its form, intensity, and frequency are exaggerated; it thus assumes the characters of a convulsive movement, inopportune and excessive; its execution is often preceded by an irresistible impulse, its suppression associated with malaise. The effect of distraction or of volitional effort is to diminish its activity; in sleep it disappears. It occurs in predisposed individuals, who usually show other indications of mental instability."

If we proceed now to the application of this carefully-worded statement, it is plain that *a tic is not a convulsion*, in the sense that the two are not identical. Cruchet has endeavoured in his thesis to base the pathological physiology of tic on researches of Monakow *à propos* of the occurrence of choreic, epileptoid, or athetotic movements after certain lesions of the cerebrospinal axis, and to find an analogy in the action of various convulsion-producing substances. His conclusion is that tic is as often spinal as cortical in origin, that it is in short a mere symptom, common to many cerebrospinal conditions. But if tic be doomed to be used indifferently for convulsion, its retention in scientific phraseology is unjustifiable. Instead of widening its significance it is preferable to restrict it, and to employ the term convulsion in its most general sense of any anomaly due to excess of muscular contraction, of whatever variety or derivation. Thus understood, tic and convulsion are far from being synonymous, inasmuch as the latter includes the former, and many other morbid phenomena.

Further, *a tic is not a spasm* According to the hypothesis advanced by Brissaud, which has the merit of explaining the facts without straining them unduly, a spasm is the result of sudden transitory irritation of any point in a reflex arc; it is a reflex act of purely spinal or bulbo-spinal origin. The focus of irritation may be situated in peripheral end-organ, on centripetal path, in medullary centre, or on centrifugal fibre. This conception of spasm may be enlarged by remembering that developmentally the gray matter of the cerebral convolutions is ectodermic, as is the skin, and capable of functioning as a sensory surface; it may be considered the end organ of an afferent path that conducts to medullary reflex centres. Hence irritation of a point on the

Rolandic cortex or on the cortico-spinal tracts is followed by a motor reaction; the sole change is in the route taken by the centripetal stimulus; the reflex centre remains bulbo-spinal, and the efferent limb of the arc is as before.

Now, though the application of the term spasm to these motor reactions is quite warranted, as for instance in Jacksonian epilepsy, it is advisable for the present to minimise the possibility of error by confining its use to conditions the result of irritation at any point on a bulbo-spinal reflex arc. Evidently then a tic is not a spasm, pure and simple. For we may suppose the reflex centre shifted from a lower to a higher level, from the bulbo-spinal region to the cortex. The modification effected by the cortex on afferent impressions is at once obvious in altered motor reactions, which appear with the stamp of cortical intervention. To this category belong the tics. If in a given motor phenomenon there is no evidence of actual or previous cortical interference, it is not a tic. If the motor reaction is consecutive to pathological irritation at any point on a bulbo-spinal reflex arc, it is a spasm. If the cortex is or has been involved in its production, it is not a spasm. Should it present, in addition to the fact of cortical participation, certain distinctive pathological features, it is a tic.

These distinctive features have been excellently summarised in an old lecture by Charcot. "However intricate and bizarre may appear the convulsive phenomena known as tics, they are not always as irregular, inco-ordinate, and contradictory, as superficial examination might lead one to believe. On the contrary, they are as a general rule systematised, seeing that in a given case they recur in an identical manner, reproducing and simultaneously exaggerating complex automatic purposive movements that are essentially physiological; they are, in a sense, the caricatures of ordinary acts and gestures. Mere repetition does not, and cannot, evolve a tic in every case. Not all who would may tic."

Such a description suffices to dispel any lingering confusion between tic and spasm, and illustrates the points on which stress is laid in Meige's definition. Tic is a psychomotor disorder, since it is clothed in the form of a convulsion, a perversion of a normal muscular contraction, and consists of a once normal act become illogical, inapposite, absurd. The imperiousness of the impulse and the peculiar satisfaction attendant on submission to it accentuate the importance of the psychical element. Of course it is readily conceivable that a spasm may be the forerunner of a tic, and in many cases no doubt a purely spasmodic reaction may determine the form and localisation which the latter will adopt; but however difficult it be to retrace the steps taken by the disease in its progress, or to satisfy one's self of the fact of cortical intervention at some stage or other, the rôle played by mental insufficiency in the genesis of tic is fundamental.

There is a danger, it may be, of emphasising the mental at the expense of the physical aspect. Magnan and his school would have it that tic is only one of the manifold expressions of degeneration, "an episodic syndrome of hereditary insanity." This is not very enlightening. Granting that all who exhibit signs of this condition are degenerates, superior or inferior as the case may be, all degenerates do not suffer from tic. Unfortunately the inclination too often is to be satisfied with the designation, and to imagine that therewith discussion terminates. Still more unfortunately, some have altogether lost sight of one of the essential characteristics of the condition, viz., the motor reaction, and have conceived the possibility of its occurrence without any tic at all. Cruchet acknowledges the existence of a "psychical tic," with no external manifestation. Such a paradox needs no refuting.

In view of the ease with which ambiguity arises, the work of analysis must be carried still further.

A tic is not a stereotyped act. It is in this connection that the inadequacy of the term "habit spasm" reveals itself. Guinon holds that tic ought not to be cited in the catalogue of diseases, since ultimately it is nothing more than a deep-rooted "bad habit." There is no call to maintain that all motor bad habits are tics, for a whole host of familiar gestures, tricks and mannerisms do not merit the name, superfluous and even detestable though they be. As long as the movement executed conserves in form the characters of a normal gesture, it is not a tic, but what Séglas calls a stereotyped act. The latter has all the appearance of a normal movement, whereas the former is a "corrupt" muscular contraction, betraying an irresistible imperiousness in its performance and requiring an agonising struggle for its repression. Concentration of the attention may diminish the intensity or even inhibit the occurrence of a tic; inversely, a simple bad habit is manifested preferably during this very concentration. The entire absence of either difficulty or distress in checking the stereotyped act indicates that the subject is psychically sound. To denominate tic as a "habit disease" is accurate enough, and we must expect to find every intermediate variety between the bad habit and the true tic, but this need not deter us from drawing the above-noted distinction.

In this brief development of the theoretical side of the question, it must have been obvious that confusion of thought has been responsible for the violent differences of opinion that characterise recent contributions, yet when we turn to consider the expression of a tic in a motor reaction, we find that here too the investigation of the subject is encircled with perplexities. Not only are the troubles of motility boundless in their diversity, and correspondingly difficult to classify; they bear also so close a resemblance to a whole series of muscular affections that the tendency is to aggregate all and sundry into one great group, under the name myoclonus, and into this chaotic farrago it is to be feared will tumble a crowd of conditions which should be studiously differentiated; tics, electric and fibrillary choreas, paramyoclonus multiplex, &c. Peril lurks in too hasty a generalisation. The scope of myoclonus is at once too inclusive and too exclusive. It may be indispensable; it is certainly not sufficient.

But the possibilities of misapprehension do not end here. On the very nature of the muscular contraction opinion is divided. That a convulsion may be either clonic or tonic is admitted, yet with singular unanimity observers have refused to recognise the existence of such a phenomenon as a "tonic tic," in spite of the instances supplied by clinical examination. So much so that a whole order of facts which, in derivation, essence, and external features, ought to be identified with the tics has been passed over in silence. Cruchet thinks that to extend the term tic to tonic contractions, such as mental torticollis, mental trismus, or permanent blepharospasm, is entirely to outstep the limits of its application. Oppenheim believes that the restriction of convulsive tic to clonic convulsions is not only warranted, but simplifies and elucidates the whole question.

To this view we conscientiously demur. The primary meaning of the word tic is not strained by applying it to a contraction of a certain duration; there is, on the contrary, historical justification for so doing. It is owing to this widening of the horizon of a tic—a proceeding strongly advocated by Brissaud and his pupils—that fresh light has been thrown on torticollis and kindred states. The interesting topic of the relations between torticollis-tic and torticollis-spasm is one which cannot be entered on in this place, but enough has been said to indicate the lines along which advance is likely to be made. In a word, recourse to the epithet convulsive is superfluous, and tic is to be taken as signifying either a clonic or a tonic contraction. The gain in conciseness is not neutralised by any loss of precision.

It has seemed fitting thus to elaborate the analytic aspect of the subject, for one cannot but be struck with the urgency of a rigorous schematisation. As it is, diagnosis will remain a matter of some complexity until the labour of analysis and synthesis alike is perfected. Many problems await solution, but that is no reason for continuing ignorant of the results already attained. It will not do to neglect the tics. A tic does not constitute a menace to life, yet the more it is repeated the more inveterate it becomes, and the greater its tendency to become generalised, while its indefinite prolongation, its association with grave mental symptoms, and its occasional termination in dementia, are excuse enough for eyeing its first premonitory exhibition with mistrust and combating it with vigour. Within recent years excellent therapeutic progress has been made by the method of re-education, and by psychomotor discipline, so that there is no need for the physician to consider himself helpless in presence of a tic, and the fact that the outlook is good is an additional plea for accuracy of diagnosis. A distinction between facial tic and facial spasm can by no means always be readily made, yet the treatment of the two is not identical. It is not at all an uncommon occurrence for chorea to degenerate into tic, and recognition of this truth is of importance, since treatment calculated to cure the one will only serve to accentuate the other. It is satisfactory to note that attention has been directed to this point in a recent lecture by Still.[1] For detailed accounts of therapeutic methods, as for the discussion of many parts of the question impracticable in a summary, reference must be made to the authorities cited, and to Meige and Feindel's book on "Les Tics et leur Traitement," a review of which has already appeared in BRAIN.[2]

S. A. K. WILSON.

---

[1] *Lancet*, December 16, 1905, p. 1,754.

[2] BRAIN, 1903, Vol. xxvi., p. 462.

Paralysis and other Diseases of the Nervous System in Childhood and Early Life. By JAMES TAYLOR, M.A., M.D., F.R.C.P., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, Queen Square; Physician to the North-Eastern Hospital for Children, and to the Moorfields Eye Hospital. London: J. and A. Churchill, 7, Great Marlborough Street, 1905.

Treatise on the Nervous Diseases of Children, for Physicians and Students. By B. SACHS, M.D., Alienist and Neurologist to Bellevue Hospital, New York. Second Edition, Revised. New York: William Wood and Company, 1905.

DR. TAYLOR is to be congratulated upon his recently published work on "Paralysis and other Diseases of the Nervous System in Childhood and Early Life." A treatise specially devoted to nervous disease in early life was a distinct desideratum, for although most writers on diseases of children deal to some extent with these disorders, it is hardly practicable to deal so fully with them in a general text-book as Dr. Taylor has been able to do here; and although in America Dr. Sachs has written on the subject, it has not, as far as we know, been treated specially by any English writer before. The title of Dr. Taylor's work disarms criticism so far as the subject matter is concerned, for he does not limit the scope of his work to nervous disease in children, he includes many diseases which are never, or hardly ever, seen in childhood, such as electrical chorea, Huntington's chorea, hereditary tabes dorsalis, and hereditary optic atrophy. Curiously enough, while many excessively rare conditions are described, there is no mention of such a peculiarly infantile disorder as spasmus nutans, which, compared with the diseases just mentioned, is almost a common affection. But more curious perhaps is the devoting of a chapter to achondroplasia in a work on nervous diseases; but here, as in other places, for instance, where some pages are occupied with a description of congenital dislocation of the hip and its diagnosis from coxa vara, the explanation evidently is that the author has in his mind the problems of diagnosis as they arise in actual practice; achondroplasia is apt to be mistaken for cretinism, coxa vara for congenital dislocation of the hip, and both these for pseudohypertrophic muscular paralysis; so that the anomaly of subjects is less than at first appears, and in reality the value of Dr. Taylor's book is enhanced by this practical view of his subject.

Dr. Sach's work is upon somewhat different lines; it is a general text-book of nervous diseases with some special reference to children: diseases such as exophthalmic goitre, tumours of the spinal cord, and disseminate sclerosis are described in detail; and many other conditions which though usually seen in the adult are occasionally seen in childhood, are discussed at full length, in some instances at much fuller length than diseases almost peculiar to children. It is noteworthy that disseminate sclerosis is stated by Sachs to be "distinctly a disease of early life," and he quotes Totzke as recording six cases under the age of 2 years, and several between 4 and 14 years of age; he mentions only one case, which began at 10 years from his own observation, and in this there was apparently no *post-mortem* verification. One may suspect that the ataxia which sometimes follows specific fevers, and which has been shown to be due to an inflammation affecting especially the cortex of the cerebellum, has sometimes been mistaken for disseminate sclerosis, and in congenital cases, such as some of those described by Totzke, which probably correspond with the condition described by Batten as congenital ataxia, there is at least a likelihood that developmental faults quite distinct from the pathological changes of disseminate sclerosis account for the symptoms.

Posterior basic meningitis is described by Dr. Taylor entirely apart from epidemic cerebro-spinal meningitis; the possibility of these being respectively sporadic and epidemic manifestations of one and the same meningeal infection is not considered. Various writers, however, including Osler, Koplik, and Sachs, have recently favoured this view, and there can be little doubt that many of the cases described as cerebro-spinal meningitis in America would be called posterior basic meningitis in England. In the use of lumbar puncture as a therapeutic method in meningitis, the author's experience agrees with that of many other observers; he says it is of no curative value in tubercular meningitis, but both here and in suppurative meningitis it may ameliorate symptoms. In the recent epidemic of cerebro-spinal meningitis in New York, it has been practised extensively, but Huber writing with reference to children says the results have been disappointing from a therapeutic point of view. Heubner, in Berlin, however, says that pain was relieved by removal of 20 to 40 c.cm. of fluid, and that many observers thought that they had obtained recovery by repeated use of lumbar puncture in these epidemic cases.

In Dr. Taylor's description of the cerebral palsies the pathology and etiology is very fully considered, and it is interesting to note how largely views have been modified as to the pathology of infantile hemiplegia within the past few years. Although in Sachs' work the polioencephalitis corticalis acuta of Strümpell is not accepted without question, and in the category of pathological conditions causing cerebral palsy in children, figures only as a suggestion at the bottom of the list, in Dr. Taylor's work the majority of cases of infantile hemiplegia are stated to be the result of an acute encephalitis, which he regards as analogous to acute anterior poliomyelitis and not improbably identical with it in its cause. Meningeal hæmorrhage, to which most writers, including Dr. Sachs, have attached much importance, is given the last place by Dr. Taylor. The view that acute anterior poliomyelitis and these cases of encephalitis are due to one and the same pathological process, raises certain difficulties; for, as this author himself points out, the spinal cord lesion is probably the result of a special and peculiar poison, and occurs occasionally in epidemic distribution; he points out also the very striking seasonal incidence of acute anterior poliomyelitis. If the two conditions, infantile hemiplegia and acute anterior poliomyelitis, be the result of the same poison whatever it may be, there should be a close correspondence in their seasonal variation, but he mentions no special seasonal incidence in the onset of infantile hemiplegia, nor so far as we are aware, has any such special incidence been recorded in that affection. Moreover, when epidemics of acute anterior poliomyelitis have occurred, there has been no special prevalence of acute encephalitis, so far as we know; Dr. Taylor, however, refers to one suggestive case, presumably that recorded by Mœbius, in which one of two children in a family developed the spinal cord affection, and the other the cerebral at the same time. He states that in "about one-third" of all cases of infantile hemiplegia the disease appears in the course of an attack of one of the specific fevers; this experience would seem to be quite exceptional. Wallenberg found 40 out of 160 to follow infectious diseases, and Osler found only 16 out of 120 to have begun in connection with, or just after, a specific fever; but the latter figures certainly, and probably Wallenberg's also, include whooping-cough, and according to Holt infantile hemiplegia occurs much more frequently with pertussis than with any of the other specific fevers. The connection with whooping-cough may be, and is, in many cases, very different from the relation of hemiplegia to other specific fevers, it is merely the result of mechanical strain rupturing vessels in the meninges or brain; moreover even in the other fevers there are often obvious causes for the occurrence of hemiplegia, quite apart from any primary infective encephalitis. Dr. Taylor refers to embolism from endocarditis in scarlet fever, and to thrombosis with marasmus; there is also a well-known tendency to thrombosis in typhoid, and the degeneration of the myocardium which occurs in diphtheria and in other specific fevers is known to cause thrombosis and embolism; until the part played by such causes in the association of hemiplegia with specific fevers can be fairly estimated, it is difficult to judge how far the theory of a primary infective encephalitis receives support from the association with the specific fevers.

In the etiology of cerebral diplegia, illness of the mother during pregnancy is mentioned as a factor of definite etiological importance; alcoholism, however, is not considered in this connection. It seems at least likely that it plays some part, whether it be present in the mother or the father; in the production of idiocy it is generally admitted to be one factor; 19·5 per cent. of the cases of idiocy at Darenth Asylum showed parental alcoholism; Koenig in a recent comparison of the etiology of cerebral palsies in children with that of idiocy showed that while 15 per cent. of cases of idiocy showed strong alcoholism in the father, no less than 23 per cent. of cases of cerebral palsy gave a similar history. The same writer regards "psychic trauma," mental shock or emotion in the mother during pregnancy, as a factor, and gives statistics showing the presence of this factor in 23 per cent. of the mothers of children with cerebral palsy.

Amaurotic family idiocy is classed by Dr. Taylor as a form of cerebral diplegia on the ground that the degeneration of nerve cells differs in extent, but not in kind, from that found in other forms of cerebral diplegia; its gradual development after birth is referred to some congenital lack of vitality in the cells, the condition to which Gowers has given the name abiotrophy. It is stated that no case has been reported in any but children of Jewish race; quite recently, however, a case has been recorded by McKee, of amaurotic family idiocy in a Gentile boy, and a careful histological examination showed generalised degeneration of nerve cells throughout the nervous system as found in other cases.

The suggestion put forward by Hirsch, and also by Russell and Kingdon, that the disease is due to some toxin acting through the mother's milk has been declared untenable by Sachs; he quotes, as an instance favouring this view, the case of a Jewish family in which the first child fed on sterilised milk was healthy, the second and third children were both breast fed, and one certainly, and probably the other also, died of this disease; but on the other hand he says that several of his cases had never been nursed by the mother, and yet had developed the disease. Sachs agrees with Taylor in accepting abiotrophy as the determining cause of the cell degeneration which is the chief feature of the disease, and he, too, notes the close resemblance between the lesions in some cases of spastic diplegia and those of amaurotic family idiocy.

Chorea is described very fully in Dr. Taylor's work, and this author inclines to the view that this disease is in the large majority of cases the result of an infective process, but that the nature of the infection is perhaps not always the same. He attaches little importance to the lesions, cortical cell changes, perivascular infiltration, capillary embolisms, &c., which have been recorded by many observers, they are to be regarded as the result of complications; the cause of the irregular movements is rather to be sought in some functional disorder of the nerve-cell itself. The recent bacteriological findings, the presence of diplococci about the small vessels of the cortex, may explain the instability of nerve-cells either by direct action of the bacteria or by the action of some bacterial toxin.

The bearing of canine chorea upon the relationship of this disease to rheumatism is not considered; the affection in dogs is said to be definitely related to distemper, which no one apparently supposes to be rheumatic; but it has yet to be proved that the so-called canine chorea corresponds to the chorea of childhood. It is stated that chorea is perhaps never met with in children under the age of 3 years; undoubtedly it is exceedingly rare under that age, but we have seen and recorded a case with articular rheumatism, endocarditis and chorea at 2 years and 10 months. To the excellent account of the treatment might be added the use of ergot, which has recently been advocated by Dr. Eustace Smith.

A chapter is devoted to stammering, which is made synonymous with stuttering; and here we confess to much sympathy with German writers, Gutzmann and others, who have drawn a sharp distinction between stuttering and stammering. For it does seem altogether incongruous to describe idioglossia and lisping as varieties of the same affection which is seen in such spasmodic co-ordination neuroses as aphthongia and the violent efforts at articulation of which Kussmaul's description is given.

In the chapter on night-terrors no mention is made of the occurrence of day-terrors, the Pavor Diurnus of Henoch, but, indeed, they are rare, so that their omission is of no great importance.

In discussing the pathology of infantile paralysis, Dr. Taylor, whilst agreeing with the observations of Dr. F. E. Batten as to the prominence of thrombosis in the morbid anatomy of acute anterior poliomyelitis, differs from that observer, and also from Dr. Sachs, in regarding this thrombosis as probably the result of a special and peculiar poison. No opinion is expressed as to whether the thrombosis is the primary change as Dr. Batten contends, or whether some inflammation occurs first with secondary thrombosis, as Drs. Head and Campbell have argued.

The theory that the disease may be dependent upon a primary affection of anterior horn-cells, with secondary changes in the interstitial tissue, is dismissed as an untenable view based upon observations taken at too late a stage of the disease to be of any value in the interpretation of its initial changes. Dr. Batten, however, whose careful observations on this disease entitle his opinion to much weight, holds that there are cases in which the primary affection is in the anterior horn cells, and that such cases form a clinical group having symptoms differing in certain well-defined respects from the commoner type of acute anterior poliomyelitis.

Dr. Taylor's work, as he himself points out, is an expression of personal opinion on subjects which have interested him; it does not claim to be an exhaustive text-book, even of the special department of disease with which it deals. Whilst, therefore, the absence of several nervous affections of childhood which one might naturally expect to find described in such a book is to be regretted, we recognise that what is lost in completeness is gained in attractiveness, for what is written *con amore*, and from personal observation, is ever more interesting than the omniscient dulness of the routine text-book. Laudatory comment is unneeded for a work such as Dr. Taylor's; "good wine needs no bush"; suffice it to say that there can be no doubt that so valuable a contribution to the subject will take a high place amongst the standard works on diseases of childhood and early life.

G. F. STILL.

Physiologische und Klinische Untersuchungen über das Gehirn. By EDUARD HITZIG. Berlin, 1904. (Hirschwald.)

"CONQUERED not by my opponents, but by all-powerful fate, which has almost entirely robbed me of my eyesight, now and for ever I lay down the knife, the pen, and the sword." These words in Professor Hitzig's preface will be read with the deepest sorrow by all neurologists. But a little while since, as Hughlings Jackson lecturer, he gained the highest honour it is in the power of the Neurological Society of Great Britain to bestow. For more than thirty years his name and his work have inspired deep respect among physiologists, and, long though his activity has lasted, his concluding works leave us with the feeling that but for this misfortune much more would have been accomplished.

The book is divided into two portions; the first contains a collection of all his earlier contributions, and these are not bare reprints, each paper is enriched by annotations and considerations of researches which have since been published by others. On re-perusing these interesting *brochures*, arranged in serial form, our admiration is immediately secured by observing how methodically Hitzig planned and set about his life's work, what pains he took first to lay down sound foundations, whereon to build up consecutive works all dependent on one another, all in regular order, and all complete. The discovery, made in the year 1870, that the surface of the brain of the dog is susceptible in places to electrical excitation, was alone sufficient to make the name of Hitzig famous. Not satisfied, however, with the results bearing on the localisation of function obtained by this means, he proceeded to supply deficiencies with a brilliant series of extirpation experiments. Next he succeeded in locating the motor area in the ape, and finally enjoyed the distinction of being the first to publish an observation on the effects of a localised lesion in man.

Many of these early papers are devoted to the localisation of the motor area, and in view of the controversies which have arisen on this subject, it is instructive to find Hitzig first stating emphatically that all the motor centres lie in the anterior central convolution, and then supplying details which agree, not absolutely but remarkably well, with those yielded by the most recent investigations. And when we consider that these observations were made when electricity had been employed for physiological purposes by this man alone, when histology had not been exploited as an adjuvant, and when apes were so scarce that one only could be procured for experiment, we see a register of the high standard of this master's work. Indeed, among pioneers in experimental neurology, there is none who has established more principles of brain physiology, none who has had less to take back as incorrect, and although he has indulged in polemics more than others, his opponents must concede that he has always met them with fair weapons.

The second half of the book consists of matter which has appeared more recently in the *Archiv für Psychiatrie und Nervenkrankheiten*. It is entirely devoted to the visual sense, and is the product of a great number of experiments on the dog, which are recorded in detail and faithfully illustrated. First, the effects of lesions of the occipital lobe on movement are discussed. In such cases Hitzig has frequently seen what he calls *Defecte der Willensenergie*, and originally thought that they denoted loss of motor impulses issuing from the destroyed parts; but now he recognises the inconstancy of the disability, and abandons it as inexplicable. Attention is next drawn to the production of visual changes by destruction of cortex away from the occipital lobe. Hitzig is assured that transitory hemiopic defects follow operations on the cortex and white substance of the sigmoid gyrus of the dog—that is, the motor area. This result has no parallel, so far as I am aware, in the cases of the human being and higher ape, and again the Professor is unable to supply a satisfactory explanation. Another interesting result was that when the temporary hemianopia caused by a lesion of the sigmoid gyrus had passed off, a lesion in the occipital region of the same side did not intensify the visual disturbance. Further, it was frequently observed that the left hemianopia to be expected from a lesion of the right occipital lobe did not occur if, previously, by lesion of the left occipital lobe, a right hemianopia had been produced. It appears correct to explain these effects by assuming that the dog is an animal in which sight is strongly represented in the primary subcortical centres.

In this part of the book Munk's conclusions are criticised with great severity. According to Hitzig partial destruction of the occipital lobe of the dog produces no permanent change in particular, the partial permanent cortical blindness of Munk does not occur. Also, the projection of the retina on the occipital lobe, as Munk describes it, is disputed; it is only admitted that a temporary quadrantic hemianopia downwards attends lesions of the anterior half of the visual area and an upward hemianopia lesions of the posterior portion. And, in Hitzig's experience, 250,000. Approximately one person in every 320 in the United States is a criminal, insane, or a pauper. With regard to heredity, which the author says is of especial potency in the evolution of crime, he quotes the statistics of Professor Poellmann, of the University of Bonn, who investigated the lives and characters of the descendants of a woman who was a confirmed drunkard. She was the direct progenitor of 834 persons, and the total cost to the State of caring for the paupers and punishing the criminals amounted to 1,206,000 dollars. The history of the "Juke family" is another case in point. The author divides criminals into four types: (1) instinctive criminals, born criminals, of which the moral imbeciles and morally insane are types; (2) criminals by impulse; (3) epileptic and insane criminals; and (4) political criminals. He refers to the efforts of Moreau, of Tours, who endeavoured to formulate the biologic effects of degenerate heredity, and himself enumerates twenty special cases of social disease. He also refers to suggestion as a cause of crime, and very rightly refers to the power of the press. The reviewer has for many years been of opinion that the publication of the morbid details of the lives of criminals in prisons causes mentally weak persons to become criminals themselves, and he is glad to find that in this opinion he is supported by Dr. Lydston. The importance of the fore-brain is admitted by the author, who expatiates on insanity in its relation to crime, the necessity for laboratory study of defectives, and the fact that suicide is more intimately associated with insanity than is any other anti-social act. Alcoholism is a common cause of crime, and lying and deceit are second nature to those addicted to the narcotic habit; but it is satisfactory to know that one of the most important factors of the prophylaxis of insanity is physical training. A chapter is devoted to anarchy in its relations to crime, and the author shows that John Wilkes Booth, Charles J. Guiteau, Garfield's assassin, and Czolgosz, the murderer of President McKinley, were all insane, and actuated by insane impulses. Sexual vice and crime occupy two chapters, and the author is very outspoken on the causes which produce prostitution. His chapter on the treatment of this sexual vice and crime contains many sensible remarks: that boys should be taught that physical perfection and early sexual indulgence are incompatible, and that "youth should be taught that to control one's appetite makes a man a king among men, but that allowing his appetites to control him makes him a slave," are not only true, but should be taught the boy by his parent or guardian. Many boys go wrong through innocence, and a masturbator should be looked upon as a curse to a school. The boy whose muscles are daily properly trained, and who takes an interest in games, is much less likely to go wrong than the loafer who has no healthy pursuits. Young girls should also be given to understand that spinsterhood is no disgrace, and far preferable to an unhappy marriage. The chapters on genius and degeneracy and physical and psychic characteristics of the criminal are well worth reading, but the review of this book has already exceeded its limit. There are numerous illustrations in it, which add much to its interest, and we can only hope that it will be read, as it should be, by all social workers.

FLETCHER BEACH.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

AT the Clinical Meeting held at 11, Chandos Street, Cavendish Square, on Thursday, October 26, at 8.30 p.m. (patients in the rooms at 8 p.m.), the following cases were shown:—

Dr. H. G. **TURNEY**.—*A case of generalised spasm, most marked in the muscles of the back, probably due to a tumour in the neighbourhood of third ventricle.*

[34] The patient is a boy, aged 16. The family history is negative; both parents are alive and well, and his one brother is a normal athletic youth. The patient's previous history is equally blank, save for childish ailments. His present condition has been coming on for about six months. Onset was vague, but less than a year ago he was bicycling and was considered to be in perfect health. He has always been a very reserved lad, but up to the average in mental development, and his mother maintains that his illness has not altered him mentally. There has been no headache or vomiting and no pain of any sort throughout.

On examination.—Patient is well grown, but emaciated. His movements are slow and difficult. The arms have a tendency to remain without support in any position in which they have been placed. The spine shows an exaggeration of the lumbar concavity and is held rigid, the back, when he stands, forming an angle of about 50° with the thighs. In the cervical region some movement is possible, but it is very limited, and the head can be turned only slightly from side to side. The stiffness of the back disappears under an anæsthetic, and an X-ray examination proved negative. The lower limbs are stiff, the legs being held at an angle with the thighs. Patient can stand with the eyes shut and can walk, though slowly, and with the limbs in the positions just described. There is optic neuritis in an early stage. No actual paralysis can be made out, and there are no changes in the electrical reactions. The deep reflexes are much increased, with ankle clonus and the

Babinski sign. There is loss of control over the bladder, the bed being constantly wet. Sensation is unaffected.

Dr. ERNEST JONES.—*Multiple bilateral contractures simulating pseudo-hypertrophic muscular paralysis. (An aberrant form of the Nageotte-Wilbonchewitch syndrome.)*

[35] F. L., male, aged 9½, out-patient under care of Dr. G. F. Still.

Family history.—Has one healthy younger sister, and no brother; none dead. Mother has had four miscarriages. Mother has three sisters and one brother well. Two sisters died in childhood. Father has seven brothers and one sister well; none dead. No similar condition to that of patient known in family.

Personal history.—Walked at 2, but never steadily. Gets pain in thighs when tired. Two years ago, when starting physical drills, it was noticed that he could not elevate his arms. Hand movements have always been clumsy. The troubles do not seem to be progressive.

Present condition.—General. Aspect unusual, owing to cheeks being very full and red, and face puffy. Chest emphysematous. Short for age. Mentally bright. Cranial nerves and fundus normal. Reflexes, sensation and sphincters natural. Muscles are everywhere weak and poorly developed, save for following exceptions:—

Posture and gait.—He stands with flexed hips and knees, and there is compensatory lordosis. This all disappears at once when he lies down. He walks with a waddling gait, limping on the right side; the joint flexion is most marked on this side. The boy gets up from the supine position in a normal manner.

Upper extremities.—(*a*) Shoulders. At the shoulder joint there is limitation both passive and active of abduction, and to a less extent of flexion and external rotation; the arm cannot be raised much above the horizontal. The scapulæ are placed further from the middle line than normal, but are not winged; the lower angles are displaced more outwards than the upper; the scapulæ move a little earlier than they should when the arm is raised, but not when the arm is lowered. The deltoids, rhomboids, lower part of trapezius and serrati magni are overdeveloped, are harder than normal. The latissimus dorsi and teres muscles are contracted on either side, especially on the right, and are of denser consistence than normal. There is no muscle atrophy either of the pectoral or other muscles.

Skiagrams show no definite joint changes.

(*b*) *Hands.*—The interphalangeal joints cannot be fully extended.

This is so in every finger and thumb, but is most marked in the terminal joints of the third and fourth fingers; the limitation is unaffected by the position of the wrists. The terminal phalanges of the fourth fingers feel thickened. Skiagrams show distinct condensation of all the terminal phalanges, and broadening, with only slight condensation of the other phalanges, so that they are bulged in the middle and not "waisted" like the metacarpals. The wrists are swollen, especially the left, where the synovial sac is felt to be large; there is undue laxity of these joints, especially of the left, which allows marked lateral mobility.

The grasp is weak, and is not strengthened by fixing the wrists. This weakness is partly, but not altogether, accounted for by limitation of flexion of the fingers, so that the hand cannot be closed even approximately. On separating the fingers, hyperextension of the metacarpo-phalangeal joints takes place; this is unaffected by the fixation or position of the wrists. Besides the interossei, the other hand muscles act fairly well.

Lower extremities.—There is slight limitation of extension and abduction; in the latter movement the abductors get tight earlier than is natural. The calf muscles are perhaps a little hard.

The skiagrams show that the head of the left femur is only ossified for a very short distance above the epiphysis. The right hip joint shows only slight defect. The acetabula are badly formed, but the cervical angles are normal. Ossification is already present in the lesser trochanters.

Dr. COLIN K. RUSSEL.—*Case of juvenile type of general paralysis of the insane.*

[36] G. B., aged 20, labourer, single, admitted under Dr. James Taylor, September 4, 1905.

Past History.—Eight months' child, always bright and active at school.

Family History.—Father had lues, and died of diabetes and tuberculosis. Mother alive and well; of her first four pregnancies three were born dead and one miscarried. The eldest living child suffers from keratitis, patient is the second.

Past Indications.—Since age of 16 he has become apathetic, with slurring and indistinct speech, cannot fix his attention, and has lost his memory. Three months ago transient palsy of right arm, with aphasia.

Present Condition.—Pupils equal, active to light and accommodation. Marked tremor about eyes, mouth and in tongue. Reflexes brisk.

Dr. F. E. BATTEN.—*(a) Ulnar neuritis, with fusiform enlargment of the nerve, occurring fifteen years after injury to the elbow.*

[37] R. S., aged 32. Eighteen years ago had a severe cog-wheel accident, with injury to the left elbow joint.

Three years ago the patient noticed numbness of the left hand, and about the same time wasting of the muscles.

At the present time there is weakness of the left hand, with atrophy of the interossei and adductor pollicis muscles. "Protopathic" sensation only is present over the distribution of the ulnar nerve. Localisation is defective, extrémes of heat and cold can only be distinguished, and touch produces the sensation of "pins and needles." There is a fusiform enlargement of the ulnar nerve as it passes over the internal condyle.

(b) Defect in upward and downward movement of the eyes. Mental impairment.

[38] T. C., aged 60. Was quite well until June 18, 1905. Went to bed well, but in the morning was found in a drowsy condition. His wife got him up and he went down stairs, but he seemed to get worse, and he was put to bed, and for the next three weeks was said to be paralysed all over. There was no headache, no vomiting, and no loss of consciousness, but his speech was defective. He gradually recovered, and was able to get about in three weeks, but his memory has always been defective since. He cannot even remember where he lives. He knows his name, and can say it indistinctly. Little or no improvement has taken place in the last few months.

His face suggests paralysis agitans, and is wanting in emotional expression. He can, however, perform all movements of the face well. The movements of the head and eyes to the right and left are normally performed. The movement of the eyes upward is defective, and on attempting this movement the lids are raised, and leave a considerable interval of sclerotic above the cornea. Downward movement of the eyes is also very defective, but if the patient is first made to converge, then the downward movement of the eyes is relatively good.

The pupils are equal and react to light and on accommodation. There is no word-deafness, no word-blindness, and no limitation of the fields of vision. Knee-jerks, active. Plantars, flexor.

Dr. FARQUHAR BUZZARD.—*A boy with some cranial nerve palsies.*

[39] W. D., aged 8.

Family and previous history good.

Present illness has an indefinite onset. For one year general listlessness and fatigue; six months getting thin, and eyes not closed during sleep; four months difficulty in swallowing; two months defective movements of eyes; one month articulation affected. No pain, headache, vomiting, or sphincter trouble.

Present condition.—Nervous and uncommunicative. General nutrition improved since admission. Taste, smell, and vision normal. Optic discs healthy. Pupils react well. Weakness of both external recti, left more than right. Slight ptosis, left more than right. Both motor and sensory functions of the fifth nerve unaffected. Marked weakness, wasting, and tremor of both sides of face, left more than right. Bilateral abductor paralysis of the larynx. Hearing good. Palatal movements deficient, but not absent. Tongue wasted, feeble, and tremulous. Swallowing slow and difficult. Limbs and trunk normal. Reflexes of the normal character.

Dr. LEONARD GUTHRIE.—*Two cases of (?) early Friedreich's disease.*

[40] (*a*) Boy, aged 4. Born healthy, except that the eyes were at first turned up, gradually assuming normal position. Has been noticed, however, to bend head on looking at objects straight in front of him, as though fixing them with the lower half of the retina.

Began to walk at 2$\frac{1}{6}$ years, but has never walked well. Seemed to be giddy, and would sometimes stagger and fall. He is a well-made, healthy-looking child, mentally backward, but not markedly deficient.

Pupils and fundi are normal. Lateral nystagmus present, and most marked on extreme deviation.

Speech babyish, drops initial consonants of words, but can repeat them correctly by imitation.

Can walk alone, but unsteadily, on wide base (there is, however, some genu valgum). Unsteadiness is much more apparent, and Rombergism is present on getting up after rest in bed.

Upper extremities.—Not ataxic. Muscles not wasted nor hypertrophied. Muscular power good, but moderate hypotonia present.

Feet considerably arched, but condition does not amount to pes cavus. No scoliosis nor kyphosis.

Sensation nowhere impaired.

Sphincters.—Nocturnal enuresis is common, but can control sphincters by day, though micturition is frequent.

Reflexes.—Knee-jerks and Achilles jerks are absent. Plantar response active, but indeterminate, sometimes flexor, sometimes extensor in type. Arm tendon-jerks not obtained. Abdominal and cremasteric reflexes brisk.

Family History.—Phthisis on father's side. Mother had serious attack of pneumonia when five months pregnant, and was hardly convalescent when patient was born.

A first cousin (paternal), a girl, aged 14, did not walk until past 3 years old, and has always been unsteady and apt to stumble and fall. There is some spastic inco-ordination of the right leg. The knee-jerks are exaggerated, ankle clonus absent. No deformity of feet, but slight scoliosis present. Eyes, speech, sensation, and sphincters normal. She is dull mentally, has a large (?) hydrocephalic head, and undersized body.

[41] (*b*) Boy, aged 11½. Healthy when born. Has always been weak on his legs and unsteady in walking.

Eighteen months ago fell off van and sustained "slight fracture of the skull with concussion of brain." Recovered entirely in a month, but weakness of the legs was as before, and during the last year has increased.

Patient is a delicate-looking anæmic boy. Intelligence good. Speech unaffected. Pupils and fundi normal. Slight nystagmus at times on lateral deviation. Muscular development fair, but slight winging of scapulæ, especially the left. No scoliosis nor deformity of feet. Slight ataxy of upper extremities. On approximating forefingers from a distance, misses contact by about an inch. Gait not ataxic, but has difficulty in walking 8-inch path. (This has improved after practice.)

Slight Rombergism present.

Knee-jerks and Achilles jerks absent. Arm-jerks and plantar reflexes unobtained. Sensation unimpaired. Muscular sense unaffected. Sphincter action normal.

Family History.—Unimportant.

Dr. Collier.—*A case of acute encephalitis resulting in partial left hemiplegia, and complete loss of vision. Optic discs healthy. Pupil reactions normal.*

[42] A child, aged 2½ years, was suddenly seized with head retraction and left hemiplegia without loss of consciousness. During the following week she had three attacks of general convulsions, most severe in the region of the face. She did not regain consciousness after the last of these attacks, but lay in an unconscious state with marked head retraction for five weeks, after which time she speedily regained consciousness and intelligence. The hemiplegia slowly improved, and eight months later she could stand, walk and use the arm a little. She has been absolutely blind ever since regaining consciousness, eight months ago. The optic discs are quite healthy, and the pupils react normally. It is suggested that the visual loss is owing to destruction of the calcarine cortex of both hemispheres.

ABSTRACT of a Paper upon "Some Investigations on Acute Rheumatic Nervous Affections," by Dr. POYNTON and Dr. ALEXANDER PAINE, read at 11, Chandos Street, Cavendish Square, on Thursday, November 23, at 8.30 p.m.

The authors, as a wish had been expressed that the entire question of the etiology of rheumatism should be open to discussion, devoted the commencement of the paper to this point. After repeating their claim that they had proved that a diplococcus was a cause of acute rheumatism, they discussed the various objections to its acceptance, and criticised their critics. They asked that the members of the Society would for the moment, at any rate, accept the view that this diplococcus was a cause of acute rheumatism, for it was the basis of the argument by which they hoped to explain the investigations they had been making upon the pathology of chorea. These investigations had commenced six years previously, and in May, 1901, in the *Lancet*, they had elaborated in some detail the view that rheumatic chorea was a result of the diplococcus infection. Granted that the diplococcus was a cause of rheumatic fever it was essential to produce evidence of its presence in the nervous system or general circulation in rheumatic chorea, at a time when the rheumatism was active, and had been the cause of death. Dana in 1894, and Apert in 1898, had found a diplococcus under these conditions, then in 1899 Westphal, Wassermann and Halkoff, isolated a diplococcus from the cerebrospinal fluid in a fatal case of pericarditis and chorea, and produced arthritis in eighty rabbits. In 1900 the authors produced choreiform movements in a rabbit with their diplococcus, and in 1901 verified the occurrence of the diplococcus in the cerebrospinal fluid in fatal rheumatism with chorea. In 1903 Beaton and Ainley Walker succeeded in isolating a diplococcus from the blood in the heart, and from the urine *ante mortem*, in three cases of chorea, and also obtained experimental results in rabbits. The authors again, at the International Congress in 1903, recorded the same experience. The experimental results included not only arthritis, but also the production of endocarditis and pericarditis.

Choreiform movements in rabbits had been observed independently by Meyer in 1902, and by Beattie with a diplococcus isolated from rheumatic arthritis in 1904.

In the present communication two further cases were recorded in which the authors had succeeded in isolating a diplococcus from the cerebrospinal fluid of children dead of fatal rheumatism with chorea. In both these cases they had advanced a step further by demonstrating the diplococci in the pia mater, thus confirming their suggestion in 1901 that this membrane would be the most likely spot in which to find them, and that the most probable localities would be in the neighbourhood of the minute blood capillaries. This view had been based upon the discovery of the diplococci in that situation in the rabbit that had developed twitching movements.

In one of these two cases they had not only succeeded in demonstrating the diplococcus in the pia mater and in isolating it from the cerebrospinal fluid, but they had also succeeded in producing on intravenous inoculations, arthritis, endocarditis and pericarditis, in a rabbit.

For this case they had been indebted to Dr. B. Lees. The patient was a boy of rheumatic parentage, aged 9, who was admitted to St. Mary's Hospital in a second attack of rheumatic fever. The symptoms had been first tonsillitis and then multiple arthritis and general chorea, finally fatal pericarditis.

The cultures from the pericardial fluid, blood and cerebrospinal fluid, had all shown the presence of the diplococcus. The morbid anatomy of chorea, they admitted, still required further investigation. A difficult point to decide was as to whether chorea was the result of a general rheumatic toxæmia, or of multiple minute local lesions. They inclined to the latter view, and compared it with the embolic theory elaborated by Hughlings Jackson. From this it differed in detail in that it seemed probable that the diplococci escaped out of the blood capillaries and produced lesions external to them. In the acute stage there resulted thrombosis, minute hæmorrhages or perivascular exudation, in the late stage of the chronic cases perivascular fibrosis. The toxins produced locally would probably cause fatty change in the nerve-cells, chorea would thus be a mild form of meningo-encephalitis and possibly meningo-myelitis. This view of the morbid anatomy was supported by the results of Reichart. The extent of their own investigations could be tabulated thus :—

(1) They had isolated and cultivated the diplococcus from the cerebrospinal fluid in four cases of fatal rheumatism, in three of which there had been chorea at the time of death.

(2) They had produced twitching movements, arthritis, endocarditis, and pericarditis by intravenous injections of this diplococcus into rabbits.

(3) They had demonstrated diplococci three times in the pia mater from cases of chorea, and once in the brain.

(4) They had demonstrated the diplococcus in the brain and pia mater of the rabbit that had shown the twitching movements.

An account was next given of a remarkable case of meningitis in a boy suffering from rheumatic fever. This boy of rheumatic parentage was admitted to University College Hospital in 1904 suffering, to all appearances, from a very ordinary attack of rheumatic fever, with multiple arthritis, acute dilatation of the heart, and early mitral endocarditis. His recovery under salicylate of sodium treatment was apparently quite a natural one, but nineteen days later, while still convalescent, he developed headache, vomiting, and moderate fever. Later he became unconscious, with convulsive movements of the limbs, and a remarkable temperature of 102·4° in the axilla and groin, and 106·4° in the rectum.

The necropsy showed early healing mitral endocarditis, a little pleurisy, and a severe non-suppurative cerebral and spinal meningitis. A minute diplococcus was present in the exudation in large numbers; it was smaller than the pneumococcus, but showed in some of the films an appearance of capsulation. Experimental inoculations produced at first rapid death in rabbits, and minute capsulated diplococci were present in the blood stream. After two months' culture in an acid medicine, the organism still retained virulence, and produced arthritis. Fully alive to the characters of the pneumococcus, they believed that this was the "diplococcus rheumaticus" in an unusually virulent condition, and thought that this was an example of true rheumatic meningitis. Finally, allusion was made to the investigations of Josué and Salomon, and also of Gandy, upon cases of cerebral hyperpyrexia in rheumatism. These investigations had pointed to that condition as being the result of an acute rheumatic toxæmia. They were led to make the following comparison between rheumatic nervous and rheumatic cardiac lesions. Mild carditis would be comparable to mild chorea, severe pericarditis to rheumatic meningitis, and acute fatal dilatation of the heart to cerebral hyperpyrexia. The paper was illustrated by microscopic slides, showing the diplococcus in the pial tissues.

At a Pathological Meeting held at 11, Chandos Street, on Thursday, December 7, at 8.30 p.m., the following communications were made :—

(43) Dr. John Turner.—*Pathological Anatomy of Idiopathic Epilepsy.*

The theory was advanced that epilepsy is a disease occurring in persons with a defectively developed nervous system associated with a morbid condition of the blood, whereby it shows a special tendency to intravascular clotting, and that the immediate cause of the fits is sudden stasis of the blood stream, resulting from the blocking of cerebral vessels by these intravascular clots.

The fits are regarded as only a symptom of the general epileptic condition.

Experimental evidence was quoted showing that sudden stasis of blood stream in the brain is capable of setting up convulsions.

In support of this thesis facts were given showing that in nearly all the cases examined appearances pointing to a defective development of the nervous system have been met with, such as:—

(α) A form of nerve-cell showing changes similar to those described as *réaction à distance.*

(β) Persistence of large numbers of subcortical nerve-cells.

Besides these, which may be looked upon as predisposing causes, it is common to meet with an *acute form of cell change* similar to that produced by ligature of the cerebral arteries in a dog.

On the part of the vascular system:—

(α) Large numbers of blood plates.

(β) Different forms of intravascular clotting, probably in large measure derived from amalgamation of the blood plates.

(γ) Small cortical hæmorrhages, which in some cases can be traced to rupture of a vessel blocked up by the aforementioned clot.

(44) Dr. J. HERBERT PARSONS.—*Brain from a case of orbital meningo-encephalocele.*

The specimen is from a child, aged 5 months, who had right orbital meningo-encephalocele associated with microphthalmia, coloboma of the optic nerve, &c. (see *Trans. Ophthalmological Soc.*, xxv., 1905). The orbit was exenterated, a large mass of brain substance being removed. This mass was found *post mortem* to spring from a large mass lying under the right frontal lobe, which it pressed upwards, and in front of the right temporo-sphenoidal lobe, which it pressed backwards. The mass is covered with pia mater and is attached to the rest of the brain only by the third nerve, which passes through it, and by blood vessels. The brain proper is apparently quite normal, apart from pressure effects. The isolated mass consists of brain substance with definite cortical lamination.

(45) Dr. Gordon Holmes.—*Specimens from a case of pseudo-hypertrophic paralysis.*

(46) Dr. Fred E. Batten.—*Two cases of acute anterior poliomyelitis.*

(47) Dr. Leonard Guthrie.—*Congenital hydrocephalus and cerebellar tumour from a youth who died, aged* 19.

Other specimens were also exhibited.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Society can obtain the Index of "Brain" for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Society the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

INDEX
TO
BRAIN:
A JOURNAL OF NEUROLOGY,
FOR
VOL. XXVIII., 1905.

INDEX OF AUTHORS.

PAGE

Ballance, C. A., and Lees, D. B.
  Case of posterior-basic meningitis, trephining, recovery ... 358

Ballet, Gilbert.
  Traité de pathologie mentale (Review) ... 73

Barnes, Stanley.
  Diffuse sarcomatous infiltration of the spinal pia mater ... 30

Barr, Martin W.
  Mental defectives (Review) ... 352

Batten, F. E.
  Ataxia in childhood ... 484
  Case of acute ataxia in a boy ... 360
  Case of tremor of right arm associated with epilepsy ... 359
  Congenital affection of sixth, seventh and twelfth cranial nuclei ... 359
  Case of defect in upward and downward movement of the eyes. Mental impairment ... 587
  Encephalitis ... 488
  Case of ulnar neuritis, with fusiform enlargement of the nerve, occurring fifteen years after injury to the elbow ... 587

Beevor, Charles E.
  Case of mesencephalic lesion ... 355
  Case of peroneal type muscular atrophy—familial type ... 354
  Case of progressive muscular atrophy, immobile pupils, exostosis on humerus ... 354

Bryant, J. H.
  Two cases of the peroneal type of family amyotrophy ... 355

Burkholder, J. F.
  The anatomy of the brain (Review) ... 90

Buzzard, E. Farquhar.
  The clinical history and *post-mortem* examination of five cases of myasthenia gravis ... 438

Buzzard, Farquhar.
  A boy with some cranial nerve palsies ... 587

PAGE

Buzzard, Thomas.
Case of mesencephalic lesion 358
Case of muscular wasting with unusual symptoms 359

Campbell, Alfred W.
Cerebral sclerosis 367

Camus, Jean, and Pagniez, Philippe.
Isolement et psychothérapie (Review) 85

Chomel, Meige, Feindel, Cruchet, &c.
Discussion sur les Tics (Review) 567

Chomel, Meige, Rudler, Dupré, &c.
Discussion sur les Tics (Review) 567

Clarke, R. H., and Horsley, Victor.
On the intrinsic fibres of the cerebellum, its nuclei and its efferent tracts 13

Collier, Dr.
A case of acute encephalitis resulting in partial left hemiplegia, and complete loss of vision. Optic discs healthy. Pupil reactions normal 589

Cruchet.
Etude critique sur le tic convulsif et son traitement gymnastique (Review) 567

Cruchet, Meige, Feindel, Rudler, Chomel, &c.
Discussion sur les Tics (Review) 567

Cruchet, Meige, Pitres, Grasset, &c.
Discussion sur les Tics (Review) 567

Dana, Charles L.
Myoclonus Multiplex (Review) 79

Dupré, Meige, Rudler, Chomel, &c.
Discussion sur les Tics (Review) 567

Feindel, Meige, Cruchet, Rudler, Chomel, &c.
Discussion sur les Tics (Review) 567

Gehuchten, A. van.
Le corps restiforme et les connexions bulbocérébelleuses (Review) 5
Les pédoncules cérébelleux supérieurs (Review) 55

Gibson, G. A.
Some hitherto undescribed symptoms in angina pectoris. ... ...

Grasset, Meige, Pitres, Cruchet, &c.
Discussion (Review) 567

Guthrie, Leonard.
Case of acute ataxy (encephalitis cerebelli) 363
Primary atrophy of optic nerves, with partial ophthalmoplegia and cerebellar gait 97
Two cases of (?) early Friedreich's disease 588

Harris, Wilfrid.
A case of chronic chorea 94
Case of transverse sacral myelitis (first sacral) 364

Head, Henry.
A case of Huntingdon's chorea 98
A case of Huntingdon's chorea 362
Case of myoclonus 362

PAGE

Head, Henry, Rivers, W. H. R., and Sherren, James.
The afferent nervous system from a new aspect 99

Head, Henry, and Sherren, James.
The consequences of injury to the peripheral nerves in man ... 116

Hitzig, Eduard.
Physiologische und klinische Untersuchungen über das Gehirn (Review) 579

Holmes, Gordon.
Case of mid-brain lesion with unilateral tremor 93

Horsley, Victor, and Clarke, R. H.
On the intrinsic fibres of the cerebellum, its nuclei and its efferent tracts 13

Horsley, Victor.
On a trigeminal-aural reflex in the rabbit 65

Hunt, J. Ramsay.
Pathology of paramyoclonus multiplex (Friedreich's type) (Review) 79

Jones, A. Ernest, M.D., M.R.C P.
The onset of hemiplegia in vascular lesions 527

Jones, Ernest.
Case of multiple bilateral contractures simulating pseudo-hypertrophic muscular paralysis. (An aberrant form of the Nageotte-Wilbon-chewitch syndrome) 585

Jones, H. Lewis.
Case of paralysis of the upper extremity after dislocation of shoulder-joint in which the distribution of paralysis was unusual 361
Case of bilateral atrophy of the intrinsic muscles of the hands in a young female 362

Lees, D. B., and Ballance, C. A.
Case of posterior basic meningitis, trephining, recovery 358

Lewandowsky, M.
Untersuchungen über die Leitungbahnen des Truncus Cerebri (Review) 556

Lugaro, E.
On unilateral hallucinations of hearing (Review) 87

Lundborg, Hermann,
Die progressive Myoklonus-epilepsia (Unverricht's Myoklonie) (Review) 79

Lydston, G. Frank.
The diseases of society and degeneracy (Review) 581

Meige, Henry.
Tics (Review) 567

Meige, Feindel, Cruchet, Rudler, Chomel, &c.
Discussion sur les Tics (Review) 567

Meige, Pitres, Cruchet, Grasset, &c.
Discussion sur les Tics (Review) 567

Meige, Rudler, Chomel, Dupré, &c.
Discussion sur les Tics (Review) 567

PAGE

Mingazzini, S.
Experimentale und pathologisch-anatomische Untersuchungen über den Verlauf einiger Bahnen des Centralnervensystems (Review) 567

Ogilvie, George.
A case of tabetic gait beginning at the age of 64 357

Oppenheim.
Bemerkungen zur Lehre vom Tic (Review) 567

Ormerod, J. A.
Case for diagnosis; possibly progressive disease of pons and peduncles 92
Case resembling Friedreich's disease, but exhibiting extensive anæsthesia 92

Pagniez, Philippe, and Camus, Jean.
Isolement et psychothérapie (Review) 85

Paine, Dr. Alexander, and Dr. Poynton.
Some investigations on acute rheumatic nervous affections (Abstract) 590

Parsons, J. Herbert.
Brain from a case of orbital meningo-encephalocele (Abstract) ... 593

Pitres, Meige, Cruchet, Grasset, &c.
Discussion (Review) 567

Pollack, Bernhard.
Die Färbetechnik für das Nervensystem (Review) 88

Poynton, Dr., and Dr. Alexander Paine.
Some investigations on acute rheumatic nervous affections ... 590

Rivers, W. H. R., Head, Henry, and Sherren, James.
The afferent nervous system from a new aspect 99

Rolleston, J. D.
A note on the condition of the tendo-Achillis jerk in diphtheria ... 68

Rudler, Meige, Feindel, Cruchet, Chomel, &c.
Discussion sur les Tics (Review) 567

Rudler, Meige, Chomel, Dupré, &c.
Discussion sur les Tics (Review) 567

Russel, Colin K.
Case of juvenile type of general paralysis of the insane 586

Sachs, B., M.D.
Treatise on the nervous diseases of children, for physicians and surgeons (Review) 573

Savill, T. D.
A case of ataxy in a woman aged 40 of twenty years' duration ... 358
Two cases of generalised tonic muscular spasm in middle-aged men, probably paralysis agitans 96

Scott, F. H., Ph.D., M.B.
On the metabolism and action of nerve cells 506

Sherren, James, Head, Henry, and Rivers, W. H. R.
The afferent nervous system from a new aspect 99

Sherren, James, and Head, Henry.
The consequences of injury to the peripheral nerves in man ... 116

PAGE

Taylor, James, M.A., M.D., F.R.C.P.
Paralysis and other diseases of the nervous system in childhood and early life (Review) 573

Thomas, Pierre.
Essai sur les altérations du cortex dans les méningites aiguës (Review) 91

Thomson, H. Campbell.
A case of tremor 357

Tuke, John Batty.
The relation of the lunacy laws to the treatment of insanity ... 1

Turner, Dr. John.
Pathological anatomy of idiopathic epilepsy (Abstract) 592

Turney, H. G.
A case of generalised spasm, most marked in the muscles of the back, probably due to a tumour in the neighbourhood of third ventricle 584

Voelcker, A. F.
A case of acute ataxy (encephalitis cerebelli) 95
Case of acute ataxy 360
Case of (?) disseminated sclerosis 361

Wilson, John Gordon.
The structure and function of the taste-buds of the larynx 339

INDEX OF SUBJECTS.

PAGE

Agenesis, lobar, with sclerosis and microgyria. Alfred W. Campbell ... 420

Amyotrophy, family, two cases of the peroneal type of. J. H. Bryant 355

Anæsthesia, case resembling Friedreich's disease, but exhibiting extensive. J. A. Ormerod 92

Angina pectoris, some hitherto undescribed symptoms in. G. A. Gibson 52

Arcuate system of cerebellum. R. H. Clarke and Victor Horsley ... 13

Arterio-sclerosis, cerebral. Alfred W. Campbell 429

Ataxy and ataxia, acute. F. E. Batten 497
  acute, a case of (encephalitis cerebelli). A. F. Voelcker 95
  acute (encephalitis cerebelli). Leonard Guthrie 363
  acute (encephalitis cerebelli). F. E. Batten 488
  case of acute, in a boy. F. E. Batten 360
  case of acute. A. F. Voelcker 360
  case of, in a woman aged 40, of twenty years' duration. T. D. Savill 358
  congenital cerebellar. F. E. Batten 485, 496
  in childhood. F. E. Batten 484
  progressive cerebellar. F. E. Batten 493, 501

Atrophy, muscular, in myasthenia gravis. E. Farquhar Buzzard ... 475
  muscular, peroneal type, familial type. C. E. Beevor 354
  of optic nerves, primary, with partial ophthalmoplegia and cerebellar gait. Leonard Guthrie 97
  progressive muscular. Immobile pupils, exostosis on humerus. C. E. Beevor 354

Aural reflex, trigeminal, in the rabbit. Victor Horsley 65

Brachial plexus, injuries to. Henry Head and James Sherren 185

Brain, anatomy of (a study of the human brain from the brain of the sheep). J. F. Burkholder (Review) 90
  from a case of orbital meningoencephalocele. J. Herbert Parsons ... 593

Cells, *see* nerve cells.

Central nervous system, experimentale und pathologisch-anatomische Untersuchungen über den Verlauf einiger Bahnen des Centralnervensystems. S. Mingazzini (Review) 557

Cerebellar ataxia, congenital. F. E. Batten 485, 496
  progressive. F. E. Batten 493, 501
  peduncles, relation of the cortex cerebelli to the. R. H. Clarke and Victor Horsley 13

Cérébelleux, supérieurs, les pédoncules. A. van Gehuchten (Review) 557

Cerebellum, cortex of the, relation of, to the cerebellar peduncles. R. H. Clarke and Victor Horsley 13
  nuclei of the, relation of the cortex cerebelli to. R. H. Clarke and Victor Horsley 13
  on the intrinsic fibres of the, its nuclei and its efferent tracts. R. H. Clarke and Victor Horsley 13
  size of the intrinsic fibres of the. R. H. Clarke and Victor Horsley 13

PAGE

Cerebral arterio-sclerosis. Alfred W. Campbell ... 429

hemiagenesis or hemisclerosis. Alfred W. Campbell ... 410

sclerosis. Alfred W. Campbell ... 367

sclerosis in epilepsy. Alfred W. Campbell ... 436

sclerosis in general paralysis of the insane. Alfred W. Campbell ... 435

sclerosis in senility. Alfred W. Campbell ... 436

sclerosis in syphilis. Alfred W. Campbell ... 437

Cerebrum, hypertrophy of. Alfred W. Campbell ... 396

Childhood and early life. Paralysis and other diseases of the nervous system in. James Taylor (Review) ... 573

ataxia in. F. E. Batten ... 484

Children, treatise on the nervous diseases of, for physicians and students. B. Sachs (Review) ... 573

Chorea, chronic, a case of. Wilfrid Harris ... 94

Huntingdon's, a case of. Henry Head ... 98, 362

Colloid sclerosis. Alfred W. Campbell ... 432

Compass test. Henry Head and James Sherren ... 228

Congenital affection of sixth, seventh and twelfth cranial nuclei. F. E. Batten ... 359

cerebellar ataxia. F. E. Batten ... 485, 496

Contractures, case of multiple bilateral, simulating pseudo-hypertrophic muscular paralysis. Ernest Jones ... 585

Cortex cerebelli, relation of, to the cerebellar peduncles. R. H. Clarke and Victor Horsley ... 13

cerebelli, relation of, to the nuclei cerebelli. R. H. Clarke and Victor Horsley ... 13

dans les méningites aiguës ; essai sur les altérations. Pierre Thomas (Review) ... 91

Cranial nerve palsies, a boy with. Farquhar Buzzard ... 587

Degeneracy, diseases of society and. G. Frank Lydston (Review) ... 581

Diphtheria, note on the condition of the tendo-Achillis jerk in. J. D. Rolleston ... 68

Diseases of society and degeneracy. G. Frank Lydston (Review) ... 581

Disseminated Sclerosis, case of (?) A. F. Voelcker ... 361

Elbow, case of ulnar neuritis, with fusiform enlargement of the nerve, occurring fifteen years after injury to. F. E. Batten ... 587

Encephalitis, case of acute, resulting in partial left hemiplegia and complete loss of vision. Dr. Collier ... 589

cerebelli, acute ataxia. F. E. Batten ... 488

Epicritic sensibility. Henry Head, W. H. R. Rivers, James Sherren 107

Epilepsy, case of tremor of right arm associated with. F. E. Batten ... 359

cerebral sclerosis in. Alfred W. Campbell ... 436

pathological anatomy of idiopathic. Dr. John Turner ... 592

Eyes, defect in upward and downward movement of. F. E. Batten ... 587

Fibres, intrinsic, of the cerebellum, size of. R. H. Clarke and Victor Horsley ... 13

Forearm, nerve supply of. Henry Head and James Sherren ... 172

Friedreich's disease, case resembling, but exhibiting extensive anæsthesia. J. A. Ormerod ... 92

disease (?), two cases of early. Leonard Guthrie ... 588

PAGE

Gait, case of primary atrophy of optic nerves, with partial ophthalmoplegia and cerebellar. Leonard Guthrie 97

General paralysis of the insane, cerebral sclerosis in. Alfred W. Campbell 435

Giant-cell sclerosis. Alfred W. Campbell 434

Hair sensibility. Henry Head and James Sherren 239

Hallucinations of hearing, on unilateral. E. Lugaro (Review) ... 87

Hand, nerve supply of palm of. Henry Head and James Sherren ... 123
  recovery of sensation after incomplete division of nerves of. Henry Head and James Sherren 163
  recovery of sensation after injury to nerves of. Henry Head and James Sherren 140

Hemiagenesis, cerebral, or hemisclerosis. Alfred W. Campbell ... 410

Hemiplegia, a case of acute encephalitis resulting in partial left, and complete loss of vision. Dr. Collier 589
  onset of, in vascular lesions. A. Ernest Jones 527

Hemisclerosis, or cerebral hemiagenesis. Alfred W. Campbell... ... 410

Huntingdon's chorea, case of. Henry Head 98, 362

Hyperalgesia. Henry Head and James Sherren 215

Hypertrophy of the Cerebrum. Alfred W. Campbell 396

Idiopathic epilepsy, pathological anatomy of. Dr. John Turner ... 592

Injury, consequences of, to peripheral nerves in man. Henry Head and James Sherren 116
  to nerves of hand, recovery of sensation after. Henry Head and James Sherren 140
  to peripheral nerves, changes in the skin that follow. Henry Head and James Sherren 251, 263

Insane, general paralysis of the, case of juvenile type of. Colin K. Russel 586

Insanity, relation of lunacy laws to treatment of. John Batty Tuke ... 1

Intrinsic fibres of cerebellum, size of. R. H. Clarke and Victor Horsley 13

Isolement et psychothérapie, traitement de l'hystérie et de la neurasthénie. Jean Camus et Philippe Pagniez (Review) 85

Larynx, structure and function of, taste-buds of. John Gordon Wilson 339

Leitungbahnen Untersuchungen über die, des Truncus Cerebri. M. Lewandowsky (Review) 556

Lesions, vascular, onset of hemiplegia in. A. Ernest Jones 527

Lobar agenesis, with sclerosis and microgyria. Alfred W. Campbell ... 420

Lunacy laws, relation of, to treatment of insanity. John Batty Tuke 1

Megalocephaly. Alfred W. Campbell 397

Meningites aiguës, essai sur les altérations du cortex dans les. Pierre Thomas (Review) 91

Meningitis, case of posterior-basic, trephining, recovery. D. Lees and C. A. Ballance 358

Meningo-encephalocele, brain from a case of orbital. J. Herbert Parsons 593

PAGE

Mental defectives. Martin W. Barr (Review) 352
impairment, defect in upward and downward movement of the eyes. F. E. Batten 587

Mesencephalic lesion, case of. C. E. Beevor 355
case of. Thomas Buzzard 358

Metabolism and action of nerve cells. F. H. Scott 506

Microgyria and sclerosis, lobar agenesis with. Alfred W. Campbell ... 420

Mid-Brain lesion, case of, with unilateral tremor. Gordon Holmes ... 93

Multiple bilateral contractures, case of, simulating pseudo-hypertrophic muscular paralysis. Ernest Jones 585

Muscular atrophy, peroneal type, familial type. C. E. Beevor 354
atrophy, progressive, immobile pupils, exostosis on humerus. C. E. Beevor 354
changes, paralysis and other, after division of peripheral nerves. Henry Head and James Sherren 275
paralysis, pseudo-hypertrophic, case of multiple bilateral contractures simulating. Ernest Jones 585
wasting, case of, with unusual symptoms. Thomas Buzzard ... 359

Myasthenia gravis, clinical history and *post-mortem* examination of five cases of. E. Farquhar Buzzard 438
gravis, morbid anatomy of. E. Farquhar Buzzard 475
gravis, muscular atrophy in. E. Farquhar Buzzard 475
gravis, pathogenesis of. E. Farquhar Buzzard... 479
gravis, sensory phenomena in. E. Farquhar Buzzard 473

Myelitis, case of transverse sacral (first sacral). Wilfrid Harris ... 364

Myoclonus, case of. Henry Head 362
multiplex. Charles L. Dana (Review) 79

Myoklonus-epilepsia, die progressive (Unverricht's Myoklonie). Hermann Lundborg (Review) 79

Nails, changes in, after injury to peripheral nerves. Henry Head and James Sherren 263

Nerve cells, action of, and hypothesis of stimulation. F. H. Scott ... 520
cells, metabolism and action of. F. H. Scott 506
cells, some constituents of, and other cells containing similar substances. F. H. Scott 506
roots, posterior, loss of sensation in the arm from division of. Henry Head and James Sherren 194
supply of forearm. Henry Head and James Sherren 172
supply of lower limb. Henry Head and James Sherren 199
supply of palm of hand. Henry Head and James Sherren 123
Nervensystem, die Färbetechnik für das. Bernhard Pollack (Review)... 88

Nerve, case of fusiform enlargement of the, with ulnar neuritis, occurring fifteen years after injury to elbow. F. E. Batten 587

Nerves, changes in the nails after injury to peripheral. Henry Head and James Sherren 263
changes in the skin that follow injury to the peripheral. Henry Head and James Sherren 251
of hand, recovery of sensation after incomplete division of. Henry Head and James Sherren 163
of hand, recovery of sensation after injury to. Henry Head and James Sherren 140
paralysis and other muscular changes after division of peripheral. Henry Head and James Sherren 275
peripheral in man, consequences of injury to the. Henry Head and James Sherren 116

PAGE

Nervous affections, some investigations on acute rheumatic. Dr. Poynton and Dr. Alexander Paine (Abstract) 590
  diseases of children, treatise for physicians and students. B. Sachs. (Review) 573
  system, afferent, from a new aspect. Henry Head, W. H. R. Rivers and James Sherren 99
  system, paralysis and other diseases of the, in childhood and early life. James Taylor (Review) 573

Neuritis, ulnar, with fusiform enlargement of the nerve, occurring fifteen years after injury to the elbow. F. E. Batten... 587

Neuroglia, development and growth of the. Alfred W. Campbell 376
  general remarks on the, in relation to cerebral sclerosis. Alfred W. Campbell... 370

Neurological Society, proceedings of 92, 354, 584

Nuclei of cerebellum, relation of cortex cerebelli to. R. H. Clarke and Victor Horsley 13
  of cerebellum, intrinsic fibres, and efferent tracts. R. H. Clarke and Victor Horsley 13
  cranial, congenital affection of sixth, seventh, and twelfth. F. E. Batten 359

Ophthalmoplegia, partial, and cerebellar gait, primary atrophy of optic nerves with. Leonard Guthrie 97

Optic nerves, primary atrophy of, with partial ophthalmoplegia and cerebellar gait. Leonard Guthrie... 97

Orbital meningo-encephalocele, brain from a case of. J. Herbert Parsons 593

Palsies, cranial nerve, a boy with. Farquhar Buzzard 587

Paralysis agitans, two cases of generalised tonic muscular spasm, probably. T. D. Savill 96

Paralysis and other diseases of the nervous system in childhood and early life. James Taylor, M.A., M.D. (Review) 573
  and other muscular changes after division of peripheral nerves. Henry Head and James Sherren 275
  general, of the insane, case of juvenile type of. Colin K. Russel ... 586
  of upper extremity after dislocation of shoulder-joint. H. Lewis Jones 361
  pseudo-hypertrophic muscular, case of multiple bilateral contractures simulating. Ernest Jones 585

Paramyoclonus, multiplex, pathology of (Friedreich's type). J. Ramsay Hunt (Review) 79

Pathology of paramyoclonus multiplex (Friedreich's type). J. Ramsay Hunt (Review) 79

Pédoncules, cérébelleux supérieurs. A. van Gehuchten (Review) ... 557

Peduncles, cerebellar, relation of the cortex cerebelli to the. R. H. Clarke and Victor Horsley 13

Peripheral nerves in man, consequences of injury to. Henry Head and James Sherren 116

Peroneal type of family amyotrophy, two cases of. J. H. Bryant ... 355
  type muscular atrophy—familial type. C. E. Beevor... 354

Pia mater, spinal, diffuse sarcomatous infiltration of. Stanley Barnes 30

Plexus, injuries to the brachial. Henry Head and James Sherren ... 185

PAGE

Pons and peduncles, progressive disease of. J. A. Ormerod ... 92

Progressive cerebellar ataxia. F. E. Batten 501
  muscular atrophy, immobile pupils, exostosis on humerus. C. E. Beevor 354

Protopathic sensibility. Henry Head, W. H. R. Rivers, James Sherren 106

Reflex, trigeminal-aural, in the rabbit. Victor Horsley 65

Reflexes, a note on the condition of the tendo-Achillis jerk in diphtheria. J. D. Rolleston 68

Rheumatic nervous affections, some investigations on acute. Dr. Poynton and Dr. Alexander Paine (Abstract) 590

Sarcomatous infiltration, diffuse, of the spinal pia mater. Stanley Barnes 30

Sclerosis and Microgyria, lobar agenesis with. Alfred W. Campbell ... 421
  cerebral. Alfred W. Campbell 367
  colloid. Alfred W. Campbell 432
  giant cell. Alfred W. Campbell 434
  tuberose. Alfred W. Campbell 382

Senility, cerebral sclerosis in. Alfred W. Campbell 436

Sensation, loss of, in arm from division of posterior nerve roots. Henry Head and James Sherren 194
  recovery of, after incomplete division of nerves of hand. Henry Head and James Sherren 163
  recovery of, after injury to nerves of hand. Henry Head and James Sherren 140

Sensations of temperature. Henry Head and James Sherren 224

Sensibility, deep. Henry Head and James Sherren 214
  of the hairs. Henry Head and James Sherren... 230

Sensory changes in angina pectoris. G. A. Gibson 52
  phenomena in myasthenia gravis. E. Farquhar Buzzard 473

Skin, changes in, that follow injury to peripheral nerves. Henry Head and James Sherren 251

Spasm, case of generalised, probably due to a tumour in the neighbourhood of third ventricle. H. G. Turney 584
  two cases of generalised tonic muscular, probably paralysis agitans. T. D. Savill 96

Spinal pia mater, diffuse sarcomatous infiltration of. Stanley Barnes 30

Syphilis, cerebral sclerosis in. Alfred W. Campbell 437

Tabetic gait, beginning at the age of 64, case of. George Ogilvie ... 357

Taste-buds of larynx, structure and function of. John Gordon Wilson 339

Temperature, sensations of. Henry Head and James Sherren 224

Tendo-Achillis jerk in diphtheria. J. D. Rolleston 68

Test, compass. Henry Head and James Sherren 228

Tic. Bemerkungen zur Lehre vom. Oppenheim (Review) 567
  convulsif, étude critique sur le, et son traitement gymnastique. Cruchet (Review) 567

Tics. Henry Meige (Review) 567
  discussion on. Congrès des Médecins aliénistes et neurologistes de France 567

PAGE

Tracts of cerebellum, intrinsic fibres, and nuclei. R. H. Clarke and Victor Horsley 13

Tremor, case of mid-brain lesion with unilateral. Gordon Holmes .. 93
  case of. H. Campbell Thomson 357
  of right arm associated with epilepsy. F. E. Batten 359

Trigeminal-aural reflex in the rabbit. Victor Horsley 465

Truncus cerebri. Untersuchungen über die Leitungbahnen des. M. Lewandowsky (Review) 556

Tuberose sclerosis. Alfred W. Campbell 382

Tumour in the neighbourhood of the third ventricle, a case of generalised spasm due to. H. G. Turney 584

Vascular lesions, onset of hemiplegia in. A. Ernest Jones ... 527

Vision, complete loss of, acute encephalitis resulting in partial left hemiplegia and. Dr. Collier 589